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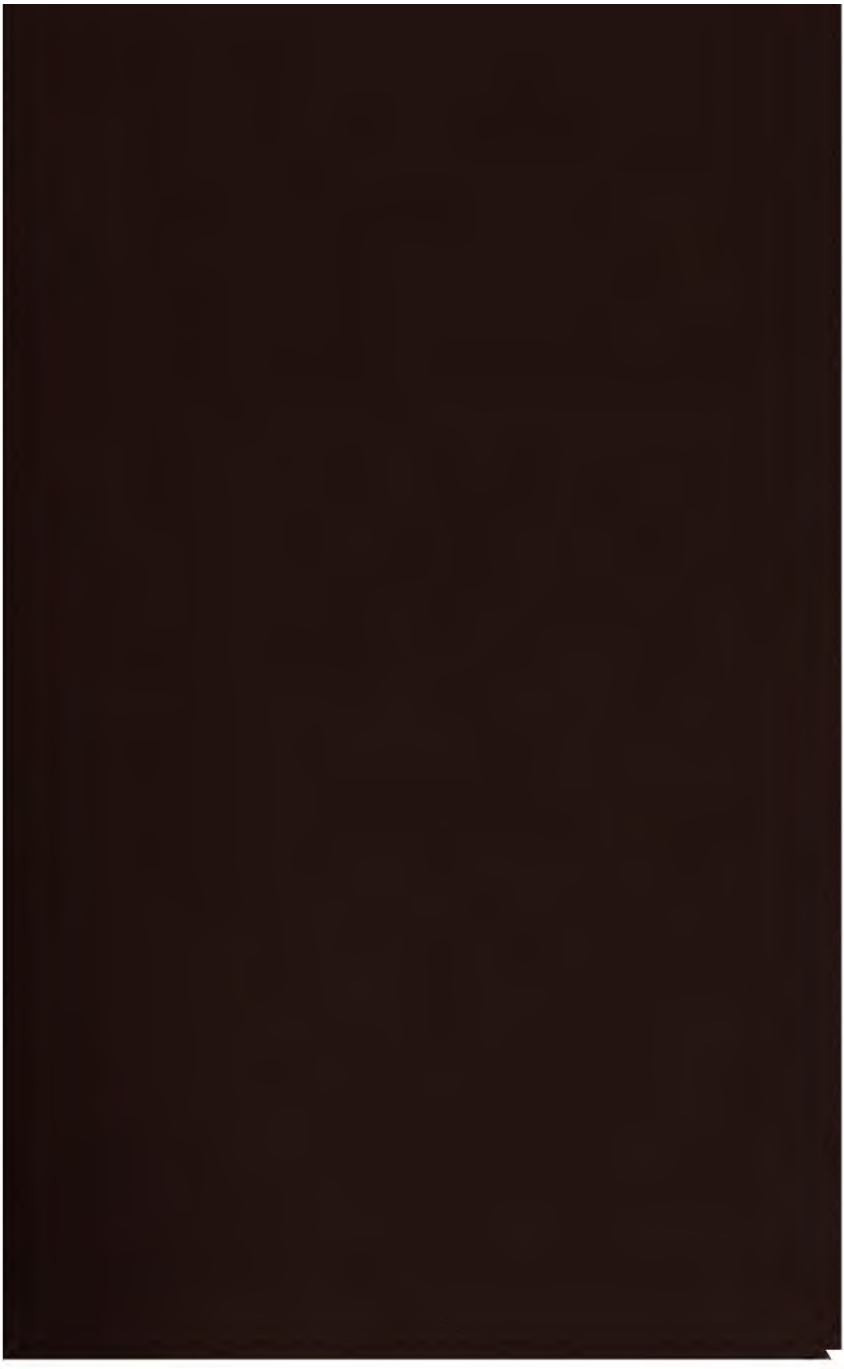

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CLINICAL LECTURES

ON

PULMONARY PHTHISIS.

FELIX VON NIEMEYER'S

CLINICAL LECTURES

ON

PULMONARY PHTHISIS.

TRANSLATED, BY PERMISSION OF THE AUTHOR,
FROM THE SECOND GERMAN EDITION,

BY

J. L. PARKE.

NEW YORK:
WILLIAM WOOD & CO., 61 WALKER STREET.
1868.

Entered according to Act of Congress, in the year one thousand eight hundred and sixty-eight,
By J. L. PARKE,
In the Clerk's Office of the District Court of the United States, for the Southern District of New York.

CLINICAL LECTURES

ON

PULMONARY PHTHISIS.

In the whole field of pathology there are no doctrines which are in such need of thorough reform as those relating to Pulmonary Phthisis. In regard to them, however, pathological anatomy has advanced far beyond clinical medicine. The expression "Pulmonary Tuberculosis" continues to be the most common term for Pulmonary Phthisis, which shows that the great majority of physicians of the present day hold fast to the standpoint of Laennec, and allow the existence of but one form of pulmonary phthisis, namely, the *tubercular*. The dangerous clause of Laennec, that "pulmonary phthisis is a constitutional disease, and that it can never be developed from an acute or chronic pneumonia, or

from a bronchial hæmorrhage," is even up to the present time proclaimed from the professor's chair as an established truth, and exercises in practice a lamentable influence on the prophylaxis and treatment of phthisis.

The dogma of Laennec, that every phthisis of the lungs is owing to a new or morbid formation, ("une espèce particulière de production accidentelle,") and that the existence of cavities in the lungs depends entirely on the softening and casting out of this morbid material, was an hypothesis which has been overthrown by the later investigations in pathological anatomy. And therewith the consequences which Laennec had deduced from that hypothesis must lose their force; and the continued assertions, that a catarrh from which a phthisis is developed must be considered not as a primary genuine catarrh, but as secondary, depending on the irritation of the lungs caused by the presence of tubercles, and that the same is true of pneumonias and pneumorrhagias which so frequently precede phthisis, prove that, in this field, practical medicine has almost entirely ignored the advances made by pathological anatomy.

The error into which Laennec and his successors fell, consisted by no means in that they held the tubercle for a morbid growth, but in that they also regarded condensations of the pulmonary tissue, which have an entirely different origin, as a development of tubercle. This confusion sprung chiefly

from the fact that the caseous metamorphosis of the originally grey and transparent tubercle was regarded as a specific peculiarity, and a sign from which could be deduced the tuberculous nature of any tissue in which it might be found. From this standpoint it was just that one should regard the extensive condensations which were found near miliary tubercle in phthisical lungs, and which at the commencement were grey and transparent, becoming later yellow and caseous, either as a diffuse development of tubercle or as an extended infiltration of the lungs with tuberculous matter. But we can no longer regard the mere fact of these diffuse condensations of the lung becoming yellow and caseous as an evidence of their tuberculous nature, especially since the pathological anatomists, and among them Virchow, have shown that formations of the most different kind, having not the slightest connection with tubercle—as, for example, old cancerous masses, lymphatic glands swollen by a hyperplasia of cells, hæmorrhagical infarctions, abscesses, &c.—undergo exactly the same caseous transformation. And therefore the whole doctrine of Laennec concerning the infiltration loses its very foundation. From the present position of science there is only one kind of tubercle, namely, the miliary tubercle, as there is only one form of tuberculosis, namely, the miliary tuberculosis, and *everything which, since the time of Laennec, has been regarded as infiltrated*

*tuberculosis of the lung, is the product of chronic and especially catarrhal pneumonia.**

It is probable that the expressions "tuberculization," "infiltrated tuberculosis," "tubercular infiltration," which have caused great confusion, and against which I contended even years ago, will finally be dropped.

But not only the doctrines of the infiltrated tuberculosis, but also those of the miliary tuberculosis have suffered from the advances made in pathological anatomy. Pathologists have become convinced that many formations which impose themselves on us at first glance as miliary granulations, and which

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* Owing to the frequent reference made by Niemeyer to the different forms of inflammations of the lungs, it would perhaps be acceptable here to quote from his work on pathology a brief definition of the different forms given by him:

"We naturally distinguish three forms of inflammations of the lungs. I. *Croupal pneumonia*," (our common acute pneumonia.) "It represents the same process in the alveoli of the lungs which, in ordinary croup, takes place in the mucous membrane of the larynx. II. *Catarrhal pneumonia* is closely connected with the processes which we have described as laryngitis, bronchitis catarrhalis, and is accompanied by increased secretion and copious formation of young cells (pus corpuscles) without an accompanying coagulating exudation into the alveoli. Both forms of inflammation deposit their exudation on the free surface, without the pulmonary tissue itself being materially disturbed in its nutrition. III. The *third* form of pneumonia, the *interstitial*, depends, on the contrary, on an inflammation which concerns the walls of the alveoli and the connective tissue lying between the lobuli. Inasmuch as the last form, at least in man, is almost always a chronic disease, it has been designated in opposition to the previous forms, which are as a rule acute, as *Chronic pneumonia*.

formerly were regarded as tubercles, have been proved on closer investigation to be oblique sections of the bronchi, with caseous contents, or bronchi whose walls are surrounded by thickened and caseous infiltrated alveoli. If, at the autopsy, we were to avoid such grounds of mistake, we could certainly arrive at the following conclusion: *that in very many cases there is not a single tubercle in phthisical lungs*, and that the condensations and destructions of the lungs depend entirely on pneumonias which have been the cause of the induration and degeneration.

Virchow, however, who has done great service in this part of pathological anatomy, goes too far when he asserts that even the views concerning miliary tuberculosis of the lungs rest almost entirely on misunderstandings, and that almost all so-called miliary tubercles of the lungs are of bronchitic, peribronchitic, or pneumonic origin. It is by no means seldom that the same transparent grey granulations, which in acute miliary tuberculosis are scattered in great numbers throughout the lungs and most other organs, and the tuberculous nature of which we cannot doubt, are met with also in phthisical lungs. But we will also be obliged to regard as tubercles those yellow caseous masses in the lungs which at present represent miliary granulations, especially if connected with them we find also in the same organ grey miliary granulations,

and if also the grey and caseous tubercles are met with in other organs.

Evidence cannot be adduced that these caseous granulations are not tubercles, but the product of vesicular pneumonia, inasmuch as we have no criterion by which we can distinguish a caseous tubercle from a caseous miliary granulation of inflammatory origin. I repeat, then, that, with the exception of the tuberculosis of the bronchial mucous membrane, I cannot grant that in the lungs of phthisical patients tubercles are often found. On the contrary, the relation of the tubercle to the condensations and degenerations of the lungs, even in such cases in which they are present side by side, is generally quite different from that which the *schoolmen* teach as the normal or ordinary relation of tubercle to the condensations and degenerations of the lung, that is, to pulmonary phthisis. If at every autopsy of that kind we ask ourselves at what period of the disease the first development of tubercle should be placed, and if we judge with eyes not dimmed by old prejudices, we will surely arrive at the conclusion, *that in most cases the tubercles have not been developed* until late, and have complicated the pulmonary phthisis at an advanced stage. Concerning tuberculosis of the intestine and larynx it has, as is well known, long been recognized that they generally connect themselves secondarily to an already existing pulmonary phthisis. I am convinced that

the same view holds good also in most cases of tuberculosis of the lungs, and I have repeatedly had the satisfaction of receiving from former pupils accounts of patients "who, after they had suffered for years with phthisis, have become finally tuberculous." In such cases the tubercles play either no part at all or a very inferior one in the destruction of the lung. As a rule, death arrives before any considerable softening and emptying out of tubercles and tuberculous conglomerates occur. Yet, notwithstanding the presence of tubercles in the lung, this form of pulmonary phthisis cannot properly be called tubercular.

It is only in a comparatively small number of cases, in which tubercles are met with in the lungs in connection with the products of chronic pneumonias, that we can in an unprejudiced examination of the post-mortem appearances, arrive at the conclusion which corresponds to the prevailing views concerning the relation of tuberculosis of the lungs to phthisis, namely, *that the development of tubercle has preceded the pneumonic processes*, and that the existing cavities are due in part to the softening and emptying out of tubercular conglomerations, and have been gradually enlarged by the softening and throwing off of tubercles, which have continued to be formed in their walls. In this form of phthisis, which should alone be called tubercular, it appears, as Virchow has first shown, that the

development of the tubercles starts, as a rule, from the bronchial mucous membrane. Here we often see, even in the trachea and the large bronchi, granulated patches, which consist of innumerable miliary tubercles, or ulcerations with the appearances of the primary and secondary tubercular ulcerations of Rokitansky, but we find moreover in these cases in the smaller bronchi, mingled with the appearances of a purulent catarrh, small whitish and yellow granulations, and we can convince ourselves, by sections made in the right direction, that the development of tubercle has spread from the bronchus to its lateral and terminal alveoli. (See Luschka, Anatomie des Menschen, 1. Band, Pag. 309.) The groups of tubercles formed in this manner appear, according to the direction of the cut, as round or wedge-shaped conglomerations of miliary granulations, an appearance which we seldom or never find in acute miliary tuberculosis, in which the development of tubercle does not proceed from the bronchial mucous membrane. The pneumonic processes, which are met with in the tubercular phthisis, in our sense of the term, are as a rule much less extensive than in that form of phthisis resulting from chronic pneumonias, and in that other form where the secondary deposit of tubercle is found only in an advanced stage of the disease. We will return later to the fact, that, the more strikingly the high fever, rapid loss of flesh, and

great dyspnœa contrast with the small extent of condensation which physical examination discloses, so much greater is the danger, so much more justified is the suspicion of tubercular phthisis. But if the fever, loss of flesh, and dyspnœa are found to correspond to the extent of condensation of lung, then the prognosis is better, because we can hope that the patient, although he is suffering from phthisis, is not laboring under tubercular phthisis.

Inasmuch as we have made the assertion that many cases of pulmonary phthisis, during their entire course, depend upon inflammatory processes, and that in most cases in which tubercles are present in the lungs in conjunction with pneumonic products, the tuberculosis has not been developed until a late stage of the disease, let us examine more closely the pneumonic processes which lead to phthisis.

And especially must we in this place earnestly oppose the statement that a peculiar pneumonia, which is therefore to be distinguished from other pneumonic forms, exists as the foundation of the caseous infiltration and the subsequent cavities. This view of a "tuberculous or caseous pneumonia" is entirely false, and threatens us with new confusions. We can on the contrary assert positively, that *every form of pneumonia undergoes, under certain circumstances, caseous infiltration, and that in no form of pneumonia is caseous infiltration the constant and only termination.* Of course the several

forms of pneumonia offer a great difference in regard to the frequency with which the products of inflammation, instead of softening and becoming absorbed, become thickened and metamorphosed into a caseous mass. In the croupal pneumonia this termination is unusual, in acute catarrhal pneumonia it is observed quite frequently, in chronic pneumonia it is almost the *rule*.

The assertion that croupal pneumonia does occasionally lead to caseous infiltration is well founded. The extreme suddenness of the invasion of the disease, as well as the symptoms and the course of it in its first stages, especially the amount of the fever and the extension of the condensation, agree so perfectly with the well known and sharply marked features of the ordinary croupal pneumonia, that we cannot doubt their identity. In corroboration we have the fact, that sometimes we have an opportunity to observe at the necropsy gradual passages from the red and grey hepatization, to the caseous metamorphosis shown by a plainly marked, granulated, cut surface. The conditions under which the product of a croupal pneumonia undergoes the caseous metamorphosis are not known, but we must remember that this termination is not only observed in persons who have tubercles, caseous masses, or cavities in the lungs, but certainly just as frequently in individuals previously healthy, and especially in persons who suffer from an old emphysema of the lung.

The termination of acute catarrhal pneumonia in caseous infiltration of the pulmonary tissue is described in a communication of Dr. Meschede,* and lately, Bartels† and Ziemsen,‡ in two excellent articles, have given a clear and perfect representation of catarrhal pneumonia in all of its phases, and have described especially its termination in caseous infiltration. The acute catarrhal pneumonia is due to an acute catarrh of the fine bronchi, which has extended into the alveoli. In this form of pneumonia the tissue of the lungs is not thickened by an exudation rich in fibrine, but by the filling up of the alveoli with young indifferent round cells. In the most favorable cases this cellular product of inflammation undergoes the same metamorphosis which the fibrine and cells imbedded in it almost always undergo in croupal pneumonia; viz., the cells fill up with fat, degenerate, and the contents of the alveoli, rendered liquid in this manner, are reabsorbed so that they become again permeable to air. In less favorable cases the cellular elements continue to congregate in the alveoli, and the fatty metamorphosis which has begun in them remains incomplete, and the cells lose their round form and shrink, from loss of water, to irregular shapes. With these microscopical alterations corresponds the ma-

* Wiener Med. Wochenschrift, 6 Jahrg., 1856, 24–25.
† Virchow's Arch., Bd. xxi, Hft. 1 und 2.
‡ Pleuritis und Pneumonie im Kindesalter. Berlin, 1862, page 293.

croscopical change from the dull shining grey or greyish-red homogeneous condensation of the pulmonary tissue into a dull yellow caseous mass. The acute catarrhal pneumonia, which generally in the beginning is a lobular process, but often spreads itself subsequently over numerous lobuli and condenses an entire lobe, occurs especially in the course of rubeola and pertussis, and the numerous cases of death which occur as sequelæ to these diseases, and which up to the present time have been referred to tuberculosis, have their origin for the most part in *the termination of an acute catarrhal pneumonia which has arisen during the disease.* But it is not infrequent that during the course of a primary true catarrh the disease extends from the bronchi into the alveoli, and causes a more or less extensive condensation of the lung.

This form of acute catarrhal pneumonia also has a termination in caseous infiltration and rapid destruction of pulmonary tissue, causing the patients to die in a short time with the symptoms of "*phthisis florida*," or "galloping consumption." Such cases are ordinarily spoken of as an "*infiltrated tuberculosis*" which has appeared under the form or mask of a bronchial catarrh or an "influenza." This inappropriate and unscientific conception is at least *convenient*, and protects the physician from the censure that he, with the exception of discovering a dullness at the apex, has neglected

the catarrh and perhaps thereby allowed it to attack the pulmonary parenchyma. Moreover, the termination of acute catarrhal pneumonia in caseous infiltration occurs by no means exclusively in persons who have already tubercles, caseous masses, or cavities in the lungs, but also in persons with previously healthy lungs. There are cases in which the conditions, under which the caseous metamorphosis of the product of an acute catarrhal pneumonia occurs, are just as obscure as those following a croupal pneumonia. "Homines quadrati" are by no means sure that they will not "catch a cold," and, as a result therefrom, have an acute catarrhal pneumonia which may eventuate as a caseous infiltration and destruction of the lung tissue. Moreover, the very strongest children can acquire from rubeola or pertussis an acute catarrhal pneumonia, the product of which undergoes caseous metamorphosis, and so carries them off. But much more frequently than in persons with a strong and healthy constitution, do we observe this termination of an acute catarrhal pneumonia in weakly and delicate individuals, who possess but little resistance against injurious influences, and therefore easily become sick and reconvalesce but slowly. When we come to speak of the etiology of pulmonary phthisis, we will dwell longer on the reasons and signs of this vulnerability, to which people often give the suitable expression "sickly;" we will, however, mention

here that individuals with weak vulnerable constistitutions show ordinarily a striking disposition, in inflammatory disturbances of nutrition, to produce a product rich in cells, and that this peculiarity is closely connected with the disposition to caseous metamorphosis of the same products of inflammation. Since we have already made the assertion that acute catarrhal pneumonia may, even in persons of previously healthy lungs, lead to caseous infiltration, we must add, that those persons who have already previously suffered from catarrhal pneumonia resulting in condensation, and who have caseous masses or cavities in the lungs, are much more liable to have subsequent attacks of acute catarrhal pneumonia take the same termination. In the description of the different forms which phthisis can assume, we will mention cases in which the patients are attacked again and again with acute catarrhal pneumonia, and in which every new attack of this disease leads to an acute extension of the condensation and destruction of the lung, until finally the patients succumb to a last attack, or die of secondary tuberculosis.

*Chronic catarrhal pneumonia** is a disease of the most frequent occurrence, and there is much true of it which is often but wrongly asserted concerning the frequency of pulmonary tuberculosis. I

* Niemeyer does not mention this form of pneumonia in his work on Pathology.—*Translator's note.*

consider *chronic catarrhal pneumonia* the only suitable term by which to designate those condensations of the lung, which are chiefly described under the name of infiltrated tuberculosis and the gelatinous or tuberculous infiltration, and which of late are just as incorrectly designated as tuberculous or caseous pneumonia.

Those lobular infiltrations of the lung which frequently become lobar by the extension of the process, having the color and appearance of frog's spawn, and showing on section a homogeneous smooth surface, depend not only on the filling up of the alveoli with young indifferent round cells, (that is, on anatomical alterations which are characteristic of catarrhal pneumonia,) but they are due also, with few exceptions, to a chronic catarrh having a secretion rich in cells, which has extended to the finest terminations of the bronchi and thence to the alveoli. I would not lay much stress on the designation of the gelatinous pneumonia as chronic catarrhal pneumonia, if I did not believe that the proper name could not only advance the comprehension of the etiology and symptomatology of phthisis but even the prophylaxis and treatment. It is not difficult to understand why the chronic catarrhal pneumonia leads, in the great majority of cases, and much more frequently than the acute form and than the croupal pneumonia, to caseous infiltration of the pulmonary tissue.

The long and tedious course of the disease which has as a result a continual increase of cells in the alveoli, and perhaps also the aspiration of cellular elements from the smallest division of the bronchi, whereby the amount of cells in the alveoli is still more increased, effects the following purely mechanical result, viz., the cells, closely pressed together, mutually infringe upon one another, become shrunken, and undergo necrobiosis (Virchow). There are, however, certain exceptions to the rule that catarrhal pneumonia terminates in caseous infiltration. If the disease ceases before this increase and crowding together of the cellular contents has reached an extreme degree, then they can undergo a complete fatty metamorphosis and become liquified and absorbed, so that the alveoli become again permeable to air. No one can deny the fact that in certain cases more or less extensive condensations of the lungs of phthisical patients disappear entirely; but this phenomenon is not in the least surprising, if we have emancipated ourselves from the prejudices occasioned by Laennec's doctrines. Chronic catarrhal pneumonia originates partly in lungs previously healthy, partly in such as contain tubercles, indurations, old caseous masses, or cavities. In the first case it forms, for the most part, the *commencement* of the nutritive disturbances to which the tubercular phthisis is due; in the second case it materially aids the extension of the conden-

sations and destructions in the lungs. Individuals with a strong and healthy constitution possess by no means an immunity against chronic catarrhal pneumonia, but it happens less frequently in them than in weakly, vulnerable individuals that a chronic catarrh extends into the alveoli.

But the caseous infiltration of the pulmonary tissue, whether induced by the one or the other form of pneumonia, does not lead in all, or even in a majority of cases, to an *immediate degeneration of the caseous infiltrated points and the formation of cavities*. Such a termination occurs rather only under certain circumstances, or perhaps only in especially violent degrees of the disease. It is probably due to this, viz., the cells heaped up in the alveoli not only mutually infringe on one another, but exercise also a certain pressure on the surrounding tissues and their blood vessels, whereby the walls of the alveoli, being deprived of their proper nutritive fluid, die. The anæmia and necrotic destruction of the pulmonary tissue is, perhaps, also increased, in severe cases, by the proliferation of cells going out from the surface and thereby causing increased pressure upon the tissue itself. At a point further on will be described more carefully the symptoms and course of the disease, as it is seen in cases in which the caseous infiltration leads to immediate degeneration of the infiltrated substance and the formation of cavities; and we will then

show that the form of disease which has its origin in that process, corresponds to "phthisis florida," or "galloping consumption."

In case the proliferation of cells is not intense enough to lead to a considerable compression of the alveolar walls and their nutritive vessels, then the caseous masses can gradually become thickened to a greater degree, and the shrunken atrophied cells degenerate into detritus; the organic substances disappear by degrees and calcareous salts are deposited, until finally there remains behind a cretaceous mortarlike concretion. In other cases, on the contrary, the shrunken cells may be rendered liquid and capable of being reabsorbed by means of the incomplete fatty metamorphosis becoming later on complete. Now while one or the other of these further metamorphoses of the cellular elements contained in the caseous infiltration is progressing, there is taking place in the lung an energetic proliferation of connective tissue. The calcified masses become capsulated, and connective tissue occupies the spots from which the cells, having undergone fatty degeneration and liquefaction, have disappeared. The pulmonary parenchyma in such cases is not rendered again permeable to air, but is changed into a firm callous mass. Inasmuch as the gradually shrinking connective tissue occupies much less space than the healthy parenchyma which it replaces, the lung becomes decreased in volume, the

thorax sinks in, and inasmuch as this sinking in is limited, the bronchi become distended to oblong or round caverns. This is the most frequent mode of the formation of cavities in phthisis having a chronic course.

The reabsorption of the caseous masses, which have at a late stage undergone fatty degeneration and liquefaction, may be so complete, that we may find at the autopsy a lung without any residual caseous matter, interspersed with "bronchiectatic" cavities, and entirely devoid of air, merely on account of the resulting interstitial induration.

From what I have asserted in the foregoing pages, one can conclude that I neither consider chronic pneumonia as an especially dangerous disease, nor indeed pulmonary phthisis, inasmuch as the latter is due principally to the former, and in fact I make no apology in stating, that the chronic inflammatory processes which lead to the condensation of the lung and the formation of cavities, show ordinarily a decided tendency to *heal*, and that under a rational treatment, individuals with extensive condensations and large cavities may often be kept for a long time in a comfortable condition, nay, may feel themselves even comparatively well. *The greatest danger for most phthisical patients is that they may become tuberculous.*

The view, that most patients with pulmonary phthisis are not in the beginning tuberculous, but

that many of them become so in the course of the disease, stands indeed in extreme opposition to the ruling opinion, but I do not doubt but that it will gain credence, because it is the only one which corresponds to the present position of pathological anatomy, and which is based upon observations, the correctness of which is generally conceded.

The fact that tuberculosis very frequently becomes conjoined with pulmonary phthisis was known to Laennec, but he has merely construed it differently from us. Laennec expresses the result of his investigations as follows: "Il est beaucoup plus commun de trouver une excavation et quelques tubercules crus, déja avancés dans le sommet des poumons, et le reste de ces organes, encore crépitans et sains d'ailleurs, farci d'une multitude innombrable de très-petits tubercules miliaires demi-transparens, et dont presque aucun ne présente encore de point jaune central. Il est évident que ces tubercules miliaires sont le produit d'une éruption secondaire et fort postérieure à celle qui avait donné lieu aux excavations. Les résultats de l'ouverte des cadavres, comparés à ceux de l'observation des malades, m'ont convaincu, que ces éruptions secondaires se font à l'époque où les tubercules formés les premiers, commencent à se ramollir."*

* Laennec. Traité de l'Auscultation de Mediate, Paris, 1831, Tome ii, pag. 27.

The foregoing sentences contain indeed exactly what we have claimed but expressed in other words. The difference lies only in the interpretation which we give to the morbid process, by which the formation of the softened nodules and the cavities in the apex of the lung take place. Laennec referred all caseous nodules and all cavities in the lungs to a previous tuberculosis, and it was therefore natural to infer, when he found a recent miliary tuberculosis existing in conjunction with the old remains of disease, that it was a secondary eruption of tubercle. We are convinced on the contrary that the caseous infiltrations and cavities of the lungs are, with few exceptions, the products of pneumonic processes, and we must therefore designate the development of tubercle, which becomes connected with them, as a complication.

Yet the frequent occurrence of tubercles in the lungs, which contain the products of chronic inflammation, contrasts in so striking a manner with the rare occurrence of tubercles in lungs free from caseous infiltrations and cavities, that we cannot well regard that complication as accidental, but are forced to admit a causal relation between the tuberculosis and the nutritive disturbances which ordinarily precede the same. We are entirely confirmed in this causal relation, when we consider the distribution of the tubercles in the lungs of phthisical patients. If there are but few tubercles present, they

are found for the most part entirely in the neighborhood of cavities or caseous masses, and if they are scattered over the whole lung, we cannot as a rule but see that their extension has proceeded from those points, because there we find the most numerous, and, as it appears, the oldest nodules.

The frequency of this complication, and the evident dependence of the tubercular development on the morbid nutrition in the lung, has doubtless materially assisted the formation of Laennec's doctrines. It was not very difficult to draw from this condition of things, the conclusion, that the inflammatory process, and the morbid formation appearing in the form of miliary nodules, were to be considered as different degrees or stages of development of one and the same disease. Moreover, the different anatomical appearance of both of these forms of morbid nutrition was by no means opposed to this construction. If we have no hesitation in referring to one and the same constitutional disease the syphilitic inflammation, and those syphilitic growths designated gummy tumors (or nodulated syphilomata), then we should make no objections when the pneumonic process and the tuberculous formations, which are so often together, are referred in the same manner to the common source of a general constitutional disease, (disposition generale, Laennec.)

But there are other reasons, aside from the ana-

tomical difference in the two processes, which refute in a striking manner this, at the first glance, enticing theory.

We must again repeat, that not only a single specific form, but *all* forms of pneumonia, lead, though not with the same frequency, to caseous infiltration and the formation of cavities in the lung, and we must also add, that in cases having this termination, the danger of tubercular complication is exactly the same, *whether the caseous products and the cavities are the result of a croupal or a catarrhal pneumonia, and whether the latter has had an acute origin or a chronic development.* If the chronic catarrhal pneumonia—the so-called gelatinous infiltration—leads more frequently than the other forms to tuberculosis, there is no other reason for it than that its products undergo the caseous metamorphosis more frequently than the products of a croupal or an acute catarrhal pneumonia.

In addition, there comes a second highly important fact. It is, namely, that almost without exception, when a tuberculosis occurs in a lung previously healthy, and free from caseous masses and cavities, *there are present in other organs, caseous products which can be due to the most different kinds of morbid processes.*

Virchow,* who, indeed, gives less importance to

* Virchow. Die krankhaften, Geschwülste, Bd. ii, pag. 727.

this phenomenon than we do, confesses that it may well be questioned whether there can exist a miliary eruption without the pre-existence of caseous, or, as Laennec would express it, softened "mother nodules."

The answer must be, that it is exceedingly seldom, for when we look carefully, there is to be found somewhere, in most every case, a caseous nodule of old date.

It is only in a few cases, that the caseous products existing in other organs (and where there is a subsequent development of tubercles) are the result of a tuberculosis, or of a process similar to the pneumonic processes. As a rule they are either enlarged lymphatic glands, which have become swollen by cellular hyperplasia, and have undergone later caseous degeneration, or they are caseous residua of pleuritic, pericarditic, peritonitic exudations, or products of chronic inflammations of the joints, bones, &c., &c., which have become caseous. Buhl,* who continues to designate the caseous masses as tubercular substance, justly states that *every tissue and every exudation in a certain stage of retrograde formation can become tubercular substance.*

This comparatively frequent coincidence of a tubercular development in the lungs, with the pres-

* Zeitschrift für Rationelle Medicin, von Henle & Pfevfers. Neue Folge. Bd. xiii, S. 51.

ence of morbid products in other organs, can by no means be considered as accidental. Such an assertion would be directly opposed to the experience of observers, as well as by the facts presented in the previously mentioned arguments. Physicians are well aware that a pleuritic exudation lasting a long time, and becoming finally converted into caseous matter, or a traumatic inflammation of the joint, or any other accidental disease which leaves behind caseous products, are even in a previously healthy individual dangerous conditions, because in numerous cases they are followed by the development of a pulmonary tuberculosis. The designation of the caseous metamorphosis of exudations and other morbid products as "tuberculization," would, save for this experience, have long since been dropped.

If, after what we have said, we again call attention to the fact that pulmonary tuberculosis follows the repeatedly mentioned pneumonic processes only in cases in which they lead to caseous infiltration, and that the tubercular development (as was already known to Laennec)* does not occur ordinarily until the time at which the caseous nodules begin to soften, then we can now affirm that between the pulmonary tuberculosis and the morbid conditions of nutrition, which generally precede it, there is by

* See the last sentence of above quotation from Laennec.

no means a direct and immediate connection resting on the common derivation from one and the same primary disease, but only an indirect connection *arising from the caseous metamorphosis of the pneumonic processes.* But we can give this sentence a more general signification, and express the same in the following manner; *tuberculosis, in most cases, is a secondary disease arising in a manner not known to us through the influence of caseous morbid products on the organism.*

We have purposely avoided adding to this assertion (which of course also holds good in the case of secondary miliary eruption called forth by caseous tubercular masses) hypotheses concerning the manner in which the caseous metamorphosis or the presence of caseous masses induces the tubercular development, in order that the impression made by the statement, which is an undeniable matter of fact, and which has great importance in understanding the etiology and symptomatology as well as the prophylaxis of tuberculosis, may not lose any of its force.

Buhl has maintained in a very decided manner the constant dependence of miliary tuberculosis on pre-existing caseous products, but he went still farther in designating, with the same confidence, miliary tuberculosis as an infectious disease arising from the reception of "tubercle poison" into the blood, and classified it with pyemia, variola, etc.

I am convinced that it is owing to this too exclusive standpoint and the extreme deductions which the ingenious investigator drew from his facts,* that the excellent work of Buhl has not exercised the reformatory influence on the clinical ideas of pulmonary tuberculosis and phthisis which they otherwise would have done. Among Buhl's twenty-three cases with miliary tubercles, there are two in which at the autopsy no pre-existing caseous mass and no caverns were found, and the number of such cases could still be considerably increased, though it would remain small when compared with the great number of undoubtedly secondary forms.

The assertion that tuberculosis is *always* dependent on caseous morbid products is one-sided and exaggerated. The idea of the infection of the blood by the caseous products has something very enticing when we consider it in connection with the acute miliary tuberculosis which is extended over most of the organs, and which indeed presents itself entirely under the clinical form of an infectious disease, but it appears to me that it by no means applies to the tuberculosis which extends itself slowly and successively with the symptoms of a hectic fever. The objections raised by Virchow

* Buhl asserts among other things: The reverse of the clause "the miliary tuberculosis is an infectious disease" also holds good; that is, an infectious disease in an individual who is suffering with caseous masses or cavities in the lungs is a miliary tuberculosis.

against the infection-theory of Buhl I do not indeed consider tenable. Even if Buhl had been right, it did not follow that every caseous nodule should lead to tuberculosis, for neither does every ichorous seat lead to septicæmia. Moreover the reabsorption of a caseous mass by its becoming altered in nature and losing its pernicious properties through advancing fatty metamorphosis and liquefaction, or through other influences, would nullify this dictum. Similar phenomena present themselves in the case of burns and ichorous regions.

On the contrary, the peculiar kind of development previously mentioned, and the not infrequent, limited presence of tubercles in the vicinity of caseous products, appear to me to point rather to a local influence, perhaps through means of the lymphatic vessels, than to an infection of the blood. The fact that the lungs, more frequently than any other organ, become the seat of tuberculosis, and that it generally remains limited to these organs, has certainly for its reason, that the diseases of the lungs are far more likely to leave behind caseous products than the diseases of other organs, and that in most cases, though by no means in all, the tubercular development remains restricted to certain limited bounds.

The ordinary assertion, that pulmonary tuberculosis leads very frequently to a secondary tuberculosis of the intestines, rests, at least in part, on a

false interpretation of what is found at the autopsy. At numerous autopsies there are indeed found cavities in the lungs and ulcerations in the intestines, that is, *a complication of tubercular and intestinal phthisis.* It is also quite common in cases in which, besides cavities and caseous masses, miliary tubercles are present in the lung, to find that the serous membrane of the intestine is covered with tubercles at points which correspond to the ulcerations of the intestine, and then we have before us a *complication of pulmonary and intestinal tuberculosis.* But it by no means follows from this that the phthisis of the intestine is induced by the phthisis of the lung, and tuberculosis of the intestine by tuberculosis of the lung. On the contrary, in a large number of cases, the unprejudiced investigation of the morbid anatomy and due regard to the phenomena of the disease during life lead us to quite a different conclusion.

Although I am not at all inclined to give the term "scrofula" too wide a signification, and shall protest against that when I come to speak of the etiology, still I consider "*scrofulous ulceration of the intestine*" as the only suitable and justifiable designation for certain and by no means rare forms of intestinal lesions which are almost always referred to as tuberculous ulcerations of the intestine. By a scrofulous ulcer is generally understood one that has originated in inflammation and ulcera-

tion of a lymphatic gland, swollen by cellular hyperplasia, and ordinarily having undergone caseous degeneration. They occur, as is known, most frequently in the neck, where they are associated with moist exanthemata of the head or face, otorrhea, diseases of the oral mucous membrane, etc. They are distinguished by their great obstinacy, by their encroachment on the adjacent tissue, and by their irregular indented and frequently undermined edges. The intestinal ulcerations in question also originate without doubt in an *ulceration of intestinal follicles which have been swollen by cellular hyperplasia and have undergone caseous degeneration.* The swelling of these small lymphatic glands is associated with chronic catarrhs of the intestine in quite an analogous manner to the swelling of the cervical glands in connection with the moist exanthemata, etc. The obstinacy, the encroachment of the ulceration on the adjacent submucous tissue, as well as the irregular indented and undermined edges, likewise remind us of the scrofulous ulcerations which we have opportunity to observe so frequently on external parts, as for instance on the neck. If we add that the "tuberculosis of the intestine" is held to be a disease especially frequent in children, in comparison with tuberculosis of the lungs, and that in children scrofulous exanthemata, scrofulous catarrhs, (that is, as proposed by Virchow, catarrhs with considerable swelling and corresponding caseous degen-

eration of the lymphatic glands,) as well as scrofulous ulcerations, are also especially frequent, and if in addition we call attention to the fact, that the ulcerations we have mentioned often have evident stamp of a very ancient origin, that they are often partly cicatrized, and that the mesenteric glands contain as a rule cretaceous masses or firm calcareous concretions, then it must be admitted that the term "tuberculous ulcerations" is for such ulcerations very unsuitable, while the term "scrofulous ulcerations" is entirely in place. A careful examination of the patient leads in many cases to the same result at which we have arrived by the unprejudiced investigation of the anatomical conditions. Hence it appears, that the patients have frequently suffered even in their childhood with diarrhœa and abdominal pains, and that sooner or later there has been established an habitual constipation interrupted from time to time by intercurrent diarrhœas. In these cases the development and even the growth of the patient is often so retarded, that young persons of twenty years make the impression of schoolboys.

I do not of course intend to assert that all ulcerations of the intestines which are called secondary tubercular ulcerations are of this scrofulous kind, but I object to confounding the two forms, and must on that account add that an entirely recent development of tubercles is met with on the serous

covering of the old scrofulous ulcerations of the intestines just as frequently as recent miliary tubercles are found in the lungs by the side of old cavities.

This circumstance, that is, the *frequent association of a tuberculosis of the intestine with a phthisis of the intestine*, which is entirely evident to impartial eyes, strengthens also to a certain degree the justness of our opinion, that a similar relation exists in the lungs, and that "patients with pulmonary phthisis are in danger of becoming tuberculous." Those by no means rare cases, in which tubercles are found only on points of the intestinal covering corresponding to the ulcerations, and in the lungs only in the immediate neighborhood of cavities, and old caseous nodules, while not a single miliary nodule can be discovered in other parts of the body, refute the correctness at least of the general ideas of Buhl's theory, according to which miliary tuberculosis is the result of an infection of the blood with the tubercle poison formed in the caseous masses.

Most of the prevailing theories concerning the inherited or acquired disposition to pulmonary tuberculosis, and especially concerning its hereditary transmission, have only a very relative value, because the observations on which they are based refer, not to tuberculosis alone, but also to all of those processes, which, since Laennec, have been

confounded with tuberculosis. The same is true of the statistics concerning the frequency of pulmonary tuberculosis in general, its greater or less frequency under certain influences, the geographical extension of the disease, etc. On the other hand, the comprehension of the etiology of pulmonary phthisis has been materially aided by the better insight we now have of the morbid conditions of nutrition which are at its foundation, and especially by the knowledge of the relation of dependence in which tuberculosis stands to other morbid processes which precede it.

Notwithstanding all the opposing opinions, I make no apology in affirming that it is not proven that tuberculosis is inheritable, if we confine ourselves to the strict meaning of the term. We can properly speak of an inherited tuberculosis only in the case when the father or the mother were suffering with genuine tuberculosis at the time of conception, and when the child is not attacked by a disease which leads to tuberculosis, but becomes directly tuberculous. In childhood, when the occurrence of pulmonary tuberculosis is rare, there can be mentioned as an example of a proper inherited tuberculosis (as Virchow and others have done,) almost nothing but, the meningeal tuberculosis, which occurs tolerably frequently at this age. But leaving out of the question the fact, that as a rule, it is not proven that the parents of such chil-

dren were in fact suffering with tuberculosis at the time of conception, it is not often, even in the case of miliary tuberculosis of the meninges, leading as it most always does, to hydrocephalus, that we are concerned with primary development of tubercles. I can scarcely remember a case when, at the autopsy of a child, who has died from tuberculosis of the cerebral membranes and acute hydrocephalus, there were not found caseous bronchial glands or other caseous masses. Buhl expressly declares that among nine cases of acute hydrocephalus with miliary tubercles in the pia mater, there was not a single case where the cerebral tuberculosis appeared isolated, and Rokitansky gives it as a rule that acute hydrocephalus of children is associated with hypertrophic development of lymphatic glands, (scrofula,) etc.

Again, cases in which adults become tuberculous, though they be descended from notoriously tuberculous parents, are not as a rule to be regarded as evidence of the power of tuberculosis to be inherited, for here also the tuberculosis is in parent and child always the last link in the chain of those morbid nutritive conditions that have originated the tuberculosis, so that it is not the tuberculosis itself that has been inherited.

With the same confidence with which we have affirmed that *the hereditary transmission of tuberculosis is not sufficiently proven*, we must now assert,

that *there is frequently an inherited disposition to pulmonary phthisis.* But even here the power to be inherited does not reside in the disease itself, but in the weakness and vulnerability of constitution, which had either laid the foundation for the pulmonary phthisis in the parents or had been developed in them as a result of the disease. The weakness and vulnerability which is bequeathed to the children may depend in the case of the parent on other influences besides pulmonary phthisis, and therefore we hear it said that children, who are descended from parents suffering from syphilis or other exhausting diseases, or whose parents were at the time of conception already aged and decrepid, have inherited a disposition to pulmonary *tuberculosis.* It is rather more correct in such cases to say the child is born with such a disposition, than to speak of its inheriting it.

However, there are numerous exceptions to the rule, that parents, who are phthisical or weakened by disease or by advanced age, generate children with a disposition to phthisis. And moreover it occurs tolerably often, that an inborn or inherited, disposition to phthisis is under favorable circumstances destroyed. Later we will dwell upon the attainment of this result, being, as it is, one of the most important indications in the prophylaxis of phthisis.

It should not be designated as idle and impracti-

cable for me to combat the inheritable disposition to tuberculosis of the lungs, while admitting the inheritable disposition to phthisis of the lungs, for I have repeatedly seen it occur that physicians, having been convinced by me of the justness of this interpretation, have become partly freed from an exaggerated and restless anxiety concerning the future of children of phthisical mothers, and have become encouraged to act energetically in warding off the danger.

The injurious influences through which the disposition to pulmonary phthisis is acquired, or the inborn disposition is increased, are sufficiently known, and we shall not enter into their full description here. As such, however, are justly mentioned insufficient and improper nourishment, bad damp dwellings, lack of exercise and exhausting influences, such as venereal excesses, prolonged lactation, depressing mental affections, etc. But I cannot refrain from adding a few words concerning the diseases which dispose to pulmonary phthisis.

Among the valuable dissertations which have been written under the direction of Dittrich, there are to be found three excellent treatises concerning the relations of the complication of pulmonary phthisis (tuberculosis) with diabetes mellitus, cancerous affections, and with the round ulcer of the stomach.*

* Die Harnruhr, von Dr. R. Leupoldt, Erlangen, 1853. Die Combina-

Dittrich has in these communications come to the conclusion that the before-mentioned forms of disease often lay the foundation of pulmonary phthisis, just as all other diseases which are followed by tabes and premature marasmus.

Although we do not agree with the interpretation which that excellent clinical observer and pathological anatomist (prejudiced however by the theories of the "crases") has proposed, and although we cannot refer tuberculosis to a diseased condition of fibrine that has been formed in the blood by the injurious influence of the products of excessive retrogressive metamorphosis, we must however acknowledge the truth of the facts.

The injurious influence which diseases have on the constitution and thereby on the disposition to pulmonary phthisis, shows itself most frequently and in the most lasting manner in earliest childhood. It is a fortunate thing for a child to remain spared from disease, especially in the earliest years, when the development of the body proceeds with the greatest rapidity, and when through favorable or unfavorable external influences the foundation for a strong and healthy or for a weakly and delicate constitution is for the most part laid. Even vaccina can considerably reduce children not other-

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tions Verhältnisse des Krebses und der Tuberculose, von Dr. C. Martin, Erlangen, 1853. Von der Combination der Tuberculose mit dem runden Magen Geschwür, von Dr. H. Papellier, Erlangen, 1861.

wise strong, not only by the eruptive fever, but by the later purulent fever, which is never wanting, and which, according to numerous temperature investigations which I have made, becomes sometimes very high, and may leave behind the germ for a disposition to pulmonary phthisis. This fact is, as is known, often falsely interpreted by uneducated and prejudiced physicians, and has led to the surprising theory, that scrofula and tuberculosis are generated by the vaccine poison continuing its influence subsequent to the inoculation.

Although we consider this theory as irrational, and the agitation raised by its believers against vaccination in general as blameable and dangerous, still we must oppose the unconditional vaccination, especially for the first two years of life. At times when no variola epidemic exists, we even forbid the vaccination of weak and delicate children, and if no cases of variola exist in the neighborhood itself or near by, we postpone the vaccination until the constitution is strengthened, and until no disadvantage can be expected from the ordinarily very inconsiderable and brief disease induced by the vaccine.

It is easily seen that all influences which lay the foundation for the inborn as well as the acquired disposition to pulmonary phthisis, agree in this, that they hinder or destroy the normal development or maintenance of the organism. If this influence shows itself before the growth of the body is com-

pleted, we see its results stamped more or less on the entire "habitus" of the individual. While, as a rule, the height of the individual has not been retarded, the breadth has suffered much more. The skin is delicate, the subcutaneous connective tissue is destitute of fat, the muscular system not well developed, the long bones are thin, the thorax displays the well known "paralytic form" depending on the scanty nutrition of its muscles. It is quite generally said of such individuals, that they are "disposed to consumption," or that they have a "consumptive look." The polemics which even to this day are often raised against these expressions, especially the objection, that not every individual with the phthisical look becomes phthisical, shows how deeply rooted are the doctrines of Laennec, according to which tuberculosis and thereby also phthisis are developed entirely independent of the reasons we have mentioned.

Taking our views as correct, it is not at all surprising to see an individual with evident disposition to pulmonary phthisis, remain free from the disease and reach an advanced age, while another, who possesses no such disposition, becomes phthisical through external influences or through intercurrent disease, etc., and succumbs at an early period of life. We can only earnestly recommend that even an inconsiderable bronchitis in an individual with a phthisical "habitus" should be regarded as a dan-

gerous enemy, and should be treated with the utmost care.

Experience teaches that delicate, badly-nourished individuals, have, as a rule, but little power of resistance against injurious influences, and that they generally acquire disease easier and recover slower than strong and well-nourished individuals. The frequency with which the different organs are attacked by disease differs, however, according to age. While in childhood, the meninges, the larynx, the skin, the intestines, etc., are especially attacked, we find at puberty the disease of the aforementioned organs in the back ground, and in their place bronchial hæmorrhages as well as acute and chronic inflammatory processes of the lungs frequently occur.

But it is not only in this vulnerability that delicate and badly-nourished individuals are distinguished from the strong and well-nourished, but also in that *the inflammatory disturbances of nutrition occurring in these lead ordinarily to a very abundant production of indifferent and transient cells.*

Among other things, it is said that such individuals have a "badly healing skin," because inconsiderable traumatic injuries cause a decided irritation of the injured part, which gives origin to a profuse production of pus cells. This peculiarity appears to rest partly on the fact that, with the weakness, there is associated an increased irritability, and partly on the fact that inflammatory irritation in

badly-nourished and incompletely developed organs leads oftener to the formation of transient cells, than of those out of which new tissue is formed.

In recapitulation we have as our conclusions the following:

Firstly.—That the condensations and destructions of the lung, which form the anatomical foundation of pulmonary phthisis, are, as a rule, the products of pneumonic processes, and that a pneumonia is the more likely to be followed by phthisis, the more abundant the cellular elements in the alveoli are, and the longer they continue, because thereby the caseous metamorphosis of the inflammatory infiltrations is promoted.

Secondly.—That pneumonia with the termination in caseous infiltration occurs principally in delicate, badly-nourished individuals; and that this is partly due to the fact, that such individuals possess but little power of resistance, and partly, that the inflammatory disturbances of nutrition occurring in them, show a tendency to abundant production of cells, and consequently to caseous metamorphosis of the inflammatory products.

Thirdly.—That such a pneumonia does not usually occur, even in delicate and vulnerable individuals, until the age at which diseases of the lungs become more frequent, the inflammatory diseases in other organs being before that time the most common.

All of the influences which dispose to pulmonary

phthisis, from its generation by a phthisical father to the exhaustion of the body by a long and tedious disease, are rendered perfectly clear and intelligible by the foregoing deductions, the correctness of which can scarcely be doubted. And when we take them into consideration there is nothing surprising, too, in the fact, that a disposition to pulmonary phthisis stamps itself more or less clearly in the sickly and delicate "habitus" of an individual.

We must add, however, that there are certain exceptions to this rule.

It sometimes occurs that individuals, without any signs of delicateness or malnutrition, become sick very frequently and without ostensible reasons, and overcome their diseases with difficulty, while others, to all appearances delicate and also with malnutrition, possess a great resistance against injurious influences, and do not become so easily diseased, and, if affected by disease, rapidly recover. On account of these exceptions, the concrete knowledge that a considerable vulnerability in fact exists, is a more reliable sign of a disposition to phthisis than the delicate and sickly look. An individual who has suffered in childhood from attacks of croup and pseudo-croup, later on from repeated catarrhs of the bronchial mucous membrane, or nasal and bronchial hæmorrhages, pneumonias, etc., is, even if he possess a healthy color and a robust look, in danger of becoming phthisical, and must accordingly,

in attacks of apparently simple catarrhs, be watched and treated with much more care than an individual concerning whom we do not have such knowledge. We cannot in a strict point of view agree with the assertion, that the disposition to pulmonary phthisis is as a rule "*associated with*" a general disposition to inflammations, for, as we believe, it has inflammatory processes for its foundation.

We can finally explain in a few words our position in the much disputed question of the relation of scrofula to pulmonary phthisis. The *lymphatic glands* also, especially in childhood, take part very frequently in the increased vulnerability, to which as a rule are associated an increased irritability and a tendency of the inflamed tissue to abundant production of cells.

While in individuals without such a special disposition, the lymphatic glands, receiving lymph from inflamed parts, swell and perhaps become inflamed, but suppurate only in violent malignant inflammations, yet in those who do have such a disposition, any irritation of the lymphatic glands, even if caused by a slight and benign inflammation at the origin of their lymphatic vessels, is sufficient to excite in them a profuse production of cells. It does not necessarily follow that an inflammation and suppuration of the glands results, but the morbid process excited by the irritation rather remains, as a rule, limited to a simple cellular hyperplasia and

swelling from the profuse collection of normal cellular elements. These glandular tumors, (just as all morbid processes in such individuals retrograde very slowly,) display extreme obstinacy, and there occurs in numerous cases a partial or diffuse caseous degeneration of the swollen glands; indeed the more profuse the collection of cellular elements becomes, so much the more probable this degeneration.

Such individuals, whose lymphatic glands participate in the general vulnerability and the tendency of the tissue to profuse production of cells in inflammatory irritations, are designated as scrofulous.

We lay no particular weight on the fact, that in scrofulous individuals the disposition to swelling of the lymphatic glands by cellular hyperplasia is associated constantly with a general disposition to diseases, especially those of an inflammatory nature. This disposition is as a rule so pronounced, that the exciting cause of the "scrofulous ophthalmia," "scrofulous catarrhs," and other so-called scrofulous affections easily escapes observation. It often appears as if those inflammations had originated spontaneously ("of themselves," as people express it.) There is no anatomical sign by which a "scrofulous ophthalmia" can be distinguished from an exanthema or ophthalmia which is not scrofulous. It is only the dependence of those inflammatory diseases on inconsiderable injuries, their frequent return, and

their obstinacy, not to speak of the participation of the lymphatic glands, which gives us a starting point from which to conclude their scrofulous nature.

Now if the slight power of resistance and the vulnerability of scrofulous individuals have not been lost at the time when the lungs become principally subject to disease, although the frequency of moist exanthemata, obstinate diseases of the cornea, conjunctiva, etc., may have decreased, pneumonic processes may originate in them just as easily, and from just as trifling causes, as previously was the case with the exanthemata, ophthalmia, etc., and moreover these pneumonic processes have the same obstinacy which those so-called scrofulous affections exhibited, a circumstance which materially promotes their termination in caseous infiltration.

We have mentioned previously that it sometimes happens, even in children, although much more rarely than in adults, and especially in the course of rubeola and pertussis, that a catarrh extends itself from the bronchioli into the alveoli, and leads to a pneumonia which may terminate in caseous infiltration. Although this may occur chiefly in scrofulous children, we object to the adoption of the term "scrofulous pneumonia," for that sort of pneumonia, because this expression would be likely to produce confusion. On the other hand we consider Virchow's proposal to designate as "scrofulous catarrhs" those catarrhs which in scrofulous individuals lead

to a considerable cellular hyperplasia in the bronchial glands, (just as a moist exanthema of the head leads to enlargement of the cervical glands,) as highly practical and acceptable.

If we leave out of sight the rare cases in which caseous bronchial glands, becoming softened and degenerated, perforate a bronchus and lead to a peculiar form of pulmonary phthisis, then we can express the relation existing according to our view, between scrofula and pulmonary phthisis, in the following manner:

Firstly.—Adults who have been scrofulous in childhood, and in whom the vulnerability on which the scrofula depends is not extinguished, have a pronounced disposition to forms of pneumonia which terminate in caseous infiltration and phthisis.

Secondly.—In certain cases bronchial glands which are left behind in previously scrofulous individuals, lay the foundation for the development of tubercles in the lung and for tubercular phthisis.

Thirdly.—Individuals who have retained from an extinguished scrofula neither an augmented vulnerability nor caseous residua in the lymphatic glands, do not possess any greater disposition to pulmonary phthisis than persons who have never been scrofulous.

I consider the wide-spread opinion that phthisis originates independently of the so-called exciting causes, and as a result only of a "diathesis," to be as

irrational as dangerous. The circumstance that it is in flagrant opposition to the established theory that pulmonary phthisis can be called forth by external circumstances, has apparently hindered an unprejudiced judgment of the facts. Lebert, among others, says: "It is important to note, that as a rule no definite cause for the beginning of chronic pulmonary tuberculosis can be found; that, for instance, taking cold appears to be in this disease devoid of any direct influence, and this is so true (!) that in cases where we are not satisfied with the vague assertions of the patient, but examine the anamnesis exactly and carefully, we can as a rule conclude that a cough the signification of which is not certain, is not of tubercular origin if it appears clearly that it has arisen from a 'cold in the head,' angina, or in short as an acute catarrh of the mucous membrane, the result of 'taking cold;' *and the popular saying that a neglected catarrh leads to consumption, is false.*"*

How can these sentences be brought to agree with the experiences of every practising physician, or with the numerous instances in which the cough has begun on a certain day after some specially injurious circumstance, and to which the complex of symptoms of phthisis has associated itself later? It is fortunate that people in general, when they

* Lebert, Handbuch der practischen Medecin, 3tte Auflage, Bd. II, S. 130.

suppose that there is a disposition to phthisis, are more careful than would be necessary if the ideas of most physicians as to the nature of phthisis were correct.

To the exciting causes, which, in conjunction with a more or less pronounced disposition to pulmonary phthisis, can induce the disease, belong, according to our views, *all injurious influences which are followed by catarrhs of the bronchi and fluxionary hyperæmia of the lungs.*

We do not need to justify ourselves any further for this assertion, for its correctness is self-evident as soon as we have left the stand-point according to which every pulmonary phthisis was derived from a morbid formation, and have become convinced that in most cases, instead of this, a catarrhal pneumonia is the foundation of the disease. We will mention farther on the rules for action, which for the most part are reprehensibly neglected in those cases where we assume, without further investigation, that a catarrh found in the apex has been caused and is maintained by a tubercular deposit, but which, on the contrary, must be employed with the utmost rigor in cases where we fear that a simple catarrh from "taking cold" may invade the alveoli and lead to phthisis.

The origin of *hyperæmia of the lungs in immoderate physical exertions* with accelerated and forcible action of the heart, a condition long recognized,

though not sufficiently explained, is, I think, brought much nearer a physiological explanation by Diesterweg's* work, which contains a series of very valuable and striking truths, though perhaps presented in a somewhat unfortunate form. I possess among my clinical reports a series of examples in which the first intimation of a commencing phthisis followed immediately after immoderate dancing, or similar excesses, and when "taking cold" at the time was improbable. Perhaps many of the cases in which phthisis is said to have been induced by a cold drink during an overheated state, belong also here.

Besides cold and excessive physical exertion, *direct irritation of the lungs and bronchial mucous membrane by foreign bodies*, plays among the exciting causes of pulmonary phthisis an extremely important part. The frequency of the disease in certain occupations is to us easy of explanation, while it would be inexplicable if in all or only in most cases of pulmonary phthisis we had to do with a morbid deposit in the lung. In the anthracosis and siderosis pulmonum (pneumonokoniosis, Zenker,) we have as a rule a phthisis of the lungs excited by inhaled coal or iron dust.

Among the foreign bodies which lead to phthisis

*Kritische Beiträge zur Physiologie und Pathologie, mit besonder Berücksichtigung des Niemeyer'schen Lehrbuches. I. Heft. Der Lungenkreislauf. Frankfurt a. M. 1866.

by an irritation of the bronchial walls and pulmonary parenchyma, the most frequent is *the coagulated blood which has remained behind in the bronchi and alveoli after an hæmoptysis or pneumorrhagia.* From Hippocrates, whose aphorism, Ἐπὶ αἵματος ἐμέτων φθόη καί τοῦ πῦου κάταρσις ἄνα, has been so often cited, up to the time of Laennec, spitting of blood was, as is known, considered as one of the most frequent causes of phthisis. For example, Hoffmann, among others, expresses himself as follows: "Verum adhuc sunt alia phthiseos initia, maximeque hæmoptysis, ubi incaute a medentibus tractatur, aut si paullo major cruoris portio est, quæ eadem amissa fuit. Tum enim facile sanguis ex pulmonum vasculis intra vesiculas aëreas extravasatur et stasi concepta putrescit, partes vicinas corrodit ac demum sinuositates efformat, vel in nodas et tubercula coit. Et prefecto, si vel meam unius hac de re experientiam adducere licet, certa asseverare fide possum, dimidiam fere partem phthisicorum qui se curæ meæ tradiderunt, ab hæmoptisi prægressa et male curata primam mali sui originem accepisse."*

The ideas of the relation of hæmoptysis to phthisis have taken an entirely different form since Laennec. We declared that the previous views rested only on an inconsiderate application of the

* Friederici Hoffmanni opera omnia. I, iii, Genevæ, 1740, page 285.

sentence, "Post hoc, ergo propter hoc;" and that even in such cases in which the hæmoptysis precedes the cough, the expectoration, and all other symptoms of phthisis, tubercles are already present in the lung. Louis declared with the same resoluteness in regard to the question, whether an hæmoptysis which precedes the cough and expectoration, should be regarded as a forerunner of the tubercles or as the first symptom, that he would be obliged to answer that the hæmoptysis "d'une manière infiniment probable" denotes the presence of tubercles in the lungs.*

This new conception, together with the entire doctrine of pulmonary phthisis, was quickly adopted, and has ruled up to the present time. This is so much the more surprising, because the declaration of Laennec stood upon a very weak foundation and rested for the most part on theory.

Laennec announced, for example, that it could not be seen anatomically how an hæmoptysis should tend to tuberculosis, and added that he had never observed the remains of an hæmoptysis transformed into millet-formed tubercles. †

And, indeed, if the assertion that every phthisis rests on tuberculosis were correct, we would have

* Recherches Anatomico-pathologiques sur la Phthisie. Paris, 1823. Pag. 194.

† Tome ii, pag. 118.

to confess that an hæmoptysis could not well lead to phthisis, inasmuch as the blood which has been poured out certainly does not become altered to tubercle.

As soon, however, as Laennec's hypothesis that in phthisis there is always a morbid deposit was overthrown, it was necessary that a reform in the ideas of the relations between hæmorrhages of the lungs and phthisis should take place. Admitting that phthisis of the lung was chiefly induced by pneumonic processes, the question arose whether it was anatomically impossible for an hæmoptysis to lay the foundation for phthisis, and no other answer could be given, than that it would be on the contrary inexplicable, if the coagulated blood contained in the bronchi and alveoli were not able, under certain circumstances, to induce pneumonic processes and caseous infiltration, and thus lead to phthisis. We have often had the opportunity of convincing ourselves that even the walls of vessels conveying blood, suffer from inflammatory disturbances of nutrition as soon as their contents become coagulated. But I would lay no great weight on this theoretical "raisonnement," if there were not innumerable and undeniable facts which tend to prove that hæmoptysis is, indeed, one of the most frequent causes of phthisis.

I make no apology in asserting, that in most cases an hæmoptysis is followed by a more or less violent

irritation of the lung and pleura. Since my attention has been called to the occurrence of this consecutive pleuro-pneumonia, I have been able almost without exception to indicate on the third or fourth day after the hæmorrhage, a rise in the temperature and increase in the frequency of the pulse, general lassitude, more or less violent lancinating pains in the lateral regions of the thorax, and often fine subcrepitant râles, friction sounds, or a slight dullness with weakened vesicular breathing or bronchial respiration. Even in cases in which a longer time has elapsed since the hæmoptysis, it was generally easy to ascertain that in the next few days after the hæmorrhage more or less evident symptoms of an inflammatory irritation of the respiratory organs had existed.

Moreover I find in the literature numerous observations of others which confirm in a striking manner the frequency of the resulting conditions,* and it is inexplicable that their constant occurrence should have been so little observed.

I do not intend to assert, that the pneumonic processes which follow an hæmoptysis, in all or even in most cases, leave behind caseous infiltrations with termination in phthisis. On the contrary, the most frequent termination is in resolution. All of the

* A specially interesting case is mentioned by Bamberger. Würzburger Medicin. Zeitschrift, II. Band, 1861, pag. 340.

symptoms often disappear in a few days, and the patient becomes entirely convalescent.

In other cases, however, the increase in the temperature and the frequency of pulse continue, the patient remains unwell during the continuance of the fever, the slight pains in the breast persist and are, as a rule, interpreted by the patient to be rheumatic, the respiration continues to be accelerated, the patient coughs and expectorates muco-purulent sputa. Should we find in conjunction with these symptoms, covering a more or less extended region of the chest, dullness on percussion, the vesicular breathing indefinite and weakened, or bronchial, and if the patient is rapidly exhausted by the increasing fever, with exacerbations in the evening and abundant night sweats, then we must fear that the pneumonic infiltration has undergone a caseous metamorphosis, and that the patient has become phthisical. And indeed nothing but the complete prejudice caused by Laennec's doctrines can explain why in such cases there has been no hesitation in deducing the phthisis from a tuberculosis, which, according to these doctrines, has remained latent up to the time of the occurrence of the hæmoptysis. With an ordinary unprejudiced judgment, we cannot otherwise construe most of the cases, in which in previously healthy individuals a galloping phthisis follows immediately upon an hæmorrhage, than *that the blood, which has remained behind in the*

bronchi and alveoli, has led to a pneumonia with caseous metamorphosis and subsequent degeneration of the retained blood with the inflammatory products, as is asserted in plain words in the previous quotation of Hoffmann.

The following case is, in this connection, especially instructive:

F. R. Wagner, tailor, thirty-two years old, was received into the clinic January 1st, 1862. The patient claims to have been healthy in childhood, was from 1851 to 1859 in the Neapolitan service; had during that time a chancre, which was followed later by ulcerations of the pharynx, exanthemata, and swellings of the head, which breaking after a time left behind tedious suppurating sores: since the winter of 1861 he has coughed and expectorated rather copiously, has also expectorated several times small quantities of blood, and has gradually lost in flesh and become very weak.

On admission the greater portion of the skin of the head was found to be changed into a suppurating ulceration, the bone was nowhere exposed, but there were noticed at several points deep depressions surrounded by irregular walls. Patient is not able to state whether any dead bone had been removed. The penis is deformed by extensive loss of substance, numerous cervical and inguinal glands are swollen, both arches of the palate show considerable defects, and the uvula is destroyed. The

skin is delicate and white, the panniculus adiposus absent, and the muscles atrophied to an extreme degree. From the apex of the left lung as far down as the third rib percussion is somewhat dull, and the tone varies on opening and shutting the mouth. In the third intercostal space there is evident "bruit de pot félé," and over the extent of the dullness loud bronchial breathing. Percussion and auscultation normal at all other points. The right lobe of the liver extends three finger-breadths beyond the lower border of the ribs. No morbid signs on the part of the other abdominal organs, pulse 80 to 91, temperature 38 to 38·5° C., 100 to 101·5° F.

Under a copious milk diet and administration of iodide of iron and cod liver oil, during January, the patient, contrary to all expectation, improved in a surprising manner. The fever almost entirely disappeared; only two or three round muco-purulent sputa were expectorated in the course of a day. The weight of the patient increased two pounds. The ulcerations of the head made rapid advances towards healing.

On the 31st of January, the patient had retired to bed, feeling comparatively well. On the following morning we found him pale and anæmic; on his table were three spittoons filled with blood. This blood, having on its surface a light foamy layer, was in other parts almost completely coagulated to a dark, nearly black, cake. To a stranger the blood

would have appeared to have been derived from a copious venesection. The patient, however, stated that he had thrown out this enormous quantity of blood within a few minutes, and that he was convinced at the time that he would bleed to death.

During the first two days after the pneumorrhagia, the patient, although greatly exhausted, was in a comfortable condition, and the physical signs, with the exception of fine subcrepitant râles over the whole extent of the right lung, were unaltered.

On the third day the patient complained of lancinating pains in the region of the left nipple. There was felt and heard at this point a rubbing sound; the pulse was accelerated, and the temperature elevated. On the 9th of February there occurred another slight hæmoptysis, which increased the exhaustion of the patient. From this time on, the disease advanced rapidly. The sound, on percussion, became tympanitic and dull over the whole extent of the left half of the thorax. Vesicular breathing was absent. Instead of the previously scanty sputa there was now an abundance of greyish-green masses thrown out; the pulse increased to 120; the temperature also increased considerably (the discouraged patient did not permit the farther use of the thermometer); abundant night sweats began; the sensorium was affected; and on the 29th of February, that is, four weeks

after the pneumorrhagia, the patient died, with all the symptoms of phthisis florida.

The autopsy was made thirty hours after death, by Prof. Liebermeister. I take from his lengthy report the following data:

Left lung, at the apex, firmly adherent. In the left pleural cavity moderate quantity of cruentous fluid mixed with fibrinous precipitations. The upper lobe of the left lung considerably shrunken; at the summit, somewhat exteriorly and anteriorly, a cavity about the size of an egg, with putrid degenerated contents; its walls bulged out and tolerably smooth. On the inner surface of the cavity there ran a large arterial branch, the walls of which were considerably thickened. Its calibre was, indeed, somewhat contracted, but not obliterated; no opening, erosion, etc., to be found. The remaining portions of the upper lobe devoid of air, partly of dark color, resembling compressed parenchyma of the lung, at certain points caseous, at others displaying small cavities with puriform contents. The lower lobe, voluminous, tough, heavy, containing but little air, showed upon section numerous infiltrated regions, from the size of a pea to a hazelnut, which adjoined each other, sometimes touched and ran together. Between the same were found smaller and larger points where the lung contained air. These points occupied about one quarter of the whole. Towards the edges of the lung the aer-

ated portions became more extensive. The infiltrated masses showed plainly on section a more or less fine or coarse granulated look; the color was in certain places red, in others grey, in others again there was a passage from one color to the other, or both colors together were present at certain points. On the whole these masses resembled an acute croupal pneumonia in the passage from the red to the grey hepatization. In many of these small, croupous, infiltrated regions about the size of a pea, the centre was found to be of a yellowish tinge, becoming of a red or grey color toward the edges. Other points within the hepatized region were found in the condition of caseous metamorphosis. These caseous points were of different sizes, partly with sharp contours and plainly separated from the surrounding infiltrated parenchyma. On the pleural covering there were seen numerous yellow, transparent points. On section of these there appeared immediately beneath the pleural surface cavities of the size of a pea and smaller, within a caseous metamorphosed tissue, which in its turn was surrounded by grey, hepatized tissue. The cavities were filled with fluid, and showed numerous dilatations. Their walls were smooth only at certain points, and covered with diphtheritic membrane; at other points numerous shreds of tissue projected into the cavity. In some instances the cavity was found to be intersected in various direc-

tions by more or less thick and branching strings, the remnants of tissue.

The formation of these cavities was due probably to the quite recent disintegration of the caseous metamorphosed parts.

Sometimes two or more cavities were in the same caseous mass, without our being able to discover any communication. At many points, especially in the interior, there were found caseous masses of the size of a hazelnut or walnut, in which very numerous cavities of the size of a pin-head or pea, were found lying in close conjunction.

A bronchus of medium size, extending towards the external lower edge of the lung, was filled with a tolerably consistent, tough, puriform mass. This mass extended from the branching end of the bronchus upwards for about two inches. At its upper end it was not adherent to the wall; the latter was, however, opaque and somewhat diphtherically infiltrated. Lower down, where the bronchus ran through completely infiltrated pulmonary parenchyma, the plug adhered tightly to the walls, *so that the whole bronchus, together with the plug, had the appearance of a vein that had been closed by a thrombus.* The wall of the bronchus was in this region tough, and thicker than it was farther upwards where the plug was not adherent. Upon removing this plug the mucous membrane was seen to be diphtheritically infiltrated, the yel-

low masses could not be torn off without tearing the substance. The plug also extended into many of the small branches, and was there more liquid.

The anterior edge of the right lung extended from the first to the fifth rib, and to the left border of the sternum. In the upper part of the upper lobe there were found numerous tolerably deep depressions of the pleura, which on section proved to be cicatrical retractions. At this point the parenchyma was filled with blood and much colored with pigment. In the interior, a small cavity of oblong form, with crumbling caseous contents, was seen.

On section of the upper lobe, there appeared small hard nodules, corresponding to sections of bronchi having thickened walls. The middle lobe was aerated, though flabby; at many points the tissue was easily broken down, and contained a considerably diminished quantity of air. Moreover, in the middle lobes there were certain nodules, out of which could be pressed inspissated puriform masses; after which the aperture in the bronchus could be clearly seen on both sides of the section. Other nodules of about the size of a pea, plainly represented infiltrations of the tissue. The lower lobe was extremely œdematous, and contained a fair amount of blood. Although aerated at all points, still the quantity of air was diminished almost throughout its entire extent. No evident infiltra-

tion of the alveoli was here to be found. Tubercles were not found, either in the lungs or in any other organ.

The large quantity of blood thrown out in a few minutes, without any admixture of air, and especially its dark color, leave no room for doubt but that the hæmorrhage in this case had its origin in an erosion or bursting of a branch of the pulmonary artery running in the wall of the old cavity. Especially instructive are the passages from the red hepatization to the caseous infiltration and to the formation of cavities; so also, is the circumstance that the recent disease of the pulmonary parenchyma showed itself almost exclusively in the left lung, in which the hæmorrhage had taken place, and in which a degenerated thrombus was found in the bronchi.

But the pneumonic processes caused by retained blood in the bronchi and alveoli, do not in all cases cause death, with the symptoms of phthisis florida, by the rapid destruction of the pulmonary tissue, from the caseous metamorphosis of the products of inflammation.

Even here the caseous masses may (as we have previously set forth) become more and more thickened and encapsuled by a proliferation of connective tissue, or may become gradually liquefied and absorbed, while a proliferation of connective tissue fills up the cavities left by this action. This termi-

nation in *induration and contraction*, betrays itself during life by a sinking in of the corresponding portion of the thorax, with a continuation of the dullness and an absent or weakened respiratory murmur. We are permitted to expect this termination, if the fever decreases, and if the general condition of the patient improves; and this improvement often occurs after we have already begun to fear the very worst. I have observed a case in which, after obstinate hæmoptysis, which threatened life, the entire lower lobe of the left lung became infiltrated, and afterwards shrunk to such a degree, that the patient consulted me a few months later, because the thorax had become oblique, a condition due undoubtedly to the retraction of the diseased lobe and the corresponding "rétrécissement" of the chest.

As an example of this termination I shall present a short extract from a letter of one of my former assistants, who consulted me from Bona in Algeria, Dr. N. among other things, writes in this letter, dated November 29th, 1862,

"While in Rotterdam in April of last year, feeling perfectly well, and not having had the least cough, as far as I remember, for years, I was seized one morning, during a violent northeast wind by an hæmoptysis; in the afternoon I had some fever, but felt the next morning quite well. The hæmoptysis, however, continued. On the third day I re-

turned to Cologne, feeling quite well, but continuing to spit blood. At Cologne I remained in bed in perfect quiet without any further symptoms than three or four bloody sputa. After about ten days I experienced, together with a certain lassitude, rheumatic pains in the back of the neck and in the second and third intercostal spaces on the left side near the sternum. Lying on the back was difficult and painful, and cough again came on with mucous expectoration. I had now tolerably high fever, and began to look poorly and lose flesh. The physical examination gave negative results, the "rheumatism" disappeared, the fever ceased, and it appeared as if I had gone through with some acute disease. Still there remained a constant, sometimes violent pain from the second to below the third rib on the left side close to the sternum, and an inconsiderable bronchial catarrh, which did not inconvenience me by day or night, but caused in the morning three or four sputa. Careful and frequent physical examinations disclosed nothing of importance in the heart or lungs. Only by frequent comparisons Dr. L. found the respiratory sound on the left side, although normal, still somewhat diminished. I recovered just as a healthy man recovers from an acute disease, and went about six weeks after to Berlin. Here there occurred a second hæmoptysis, which lasted about fourteen days, but did not in any manner disturb my general health, and left no

catarrh behind. The physical examination made in Berlin by T. and F. disclosed even now nothing else than diminished though normal respiration, no subcrepitant râles. I remained in Berlin until Easter, 1862, feeling quite well, when during a visit from my mother I was attacked by a third hæmoptysis of about four days' duration, and which was again followed by no bad results. At Whitsuntide I had a fourth and tolerably abundant hæmoptysis, and in July a fifth, by far the worst. This lasted three to four weeks; I continually expectorated pure blood, and indeed, during a week, very considerable quantities. At the end of the second week I was reduced very low and *had violent pleuritic pain and dyspnœa, which rendered local bleeding necessary.* Even this passed away like an acute disease, *and I recovered completely, with excellent appetite up to the end of September.* This time, however, the cough which had remained behind was more considerable, and the insufficient expansion of the lung could easily be perceived on a somewhat forcible inspiration.

"The pain in the above mentioned region had remained, and *the region itself was quite flattened or concave.* At the present time *its resistance is increased, and the respiratory sound is weaker.* There occur still, from time to time, apparently rheumatic pains in the muscles which surround the upper portion of the thorax.

"In conclusion, I repeat, that during the whole

time, I have lost neither flesh nor strength, with the exception of those periods during which I had fever and violent pain. The cough has diminished, and I notice the insufficiency of the lung only when going up hill."*

Finally, it should be mentioned that an hæmoptysis may lead, not only to a destructive and indurating pneumonia, but later also, to tuberculosis, on account of the remaining caseous regions. I possess among my reports certain very striking examples of this course of the disease.

To defend myself from being misunderstood, I must mention finally, that in the foregoing pages I have referred only to that relation between hæmoptysis and pulmonary phthisis, which is of late not at all acknowledged, or too little observed, and that I by no means consider this as the only relation which exists between hæmorrhages from the air passages and pulmonary phthisis. On the contrary, I consider an hæmoptysis, especially one occurring under trifling causes, apart from the danger of coagulated blood being left behind in the alveoli, as by itself, mali ominis, because experience teaches us that a morbid fragility (hæmorrhagic diathesis) of the final branches of the bronchial artery in the bronch-

* The three cases here mentioned are to be found more fully reported in the dissertation of Dr. Bürger: Ueber das Verhältniss der Bronchial- und Lungen-Blutungen. Tübingen, 1864.

ial mucous membrane, is, as a rule, associated with a tendency to inflammatory diseases of the parenchyma of the lung, which also receives its nutrition from the same arteries.

I confess, willingly, that bronchial hæmorrhages occur during the course of pulmonary phthisis, and even in all of its stages, and far more frequently than they precede it.

My views concerning the relation between bronchial hæmorrhages and pulmonary phthisis are embraced briefly and concisely in the following statements:

Firstly.—Abundant bronchial hæmorrhages occur more frequently than is generally admitted in persons who are neither at the time of the hæmorrhage phthisical, nor become so later.

Secondly.—In many cases, abundant hæmorrhages of the bronchial mucous membrane precede the beginning of pulmonary phthisis without the existence of any genetical connection between the hæmorrhage and the disease of the pulmonary tissue. In this case, both processes have the same origin; namely, the common disposition of the patients to bronchial hæmorrhage, on the one hand, and to pulmonary phthisis on the other.

Thirdly.—Hæmorrhages of the bronchial mucous membrane precede the development of a pulmonary phthisis, and stand in direct genetical connection with it; because the bronchial hæmorrhage leads to

chronic inflammatory processes of the pulmonary tissue, with termination in its destruction.

Fourthly.—Bronchial hæmorrhages occur in already existing pulmonary phthisis still more frequently than they precede it; they appear (in rare cases, however) even at a period in which the pulmonary disease is still latent.

Fifthly.—Bronchial hæmorrhages, which occur during the course of a pulmonary phthisis, can hasten the fatal termination of this disease by inducing chronic destructive inflammatory processes.

The symptomatology of pulmonary phthisis varies according to whether the symptoms are occasioned from beginning to end by pneumonic processes, or whether the latter become complicated at a later period with tuberculosis, or finally, whether the disease begins with a tuberculosis. In most cases these three forms can be distinguished from one another with tolerable certainty.

From our present position we shall subject to a brief criticism the separate symptoms upon which the diagnosis of pulmonary phthisis is generally founded, and then investigate particularly to which of the above processes each one of these symptoms

belongs. Further on we shall endeavor to describe the course of each of *these three forms of pulmonary phthisis.*

Increased frequency of respiration occurs in all forms of pulmonary phthisis, though in a different degree, and owing to a variety of causes. A moderate increase in frequency is not always accompanied by dyspnœa.

It is very frequently the case, that patients with pulmonary phthisis in an advanced stage experience dyspnœa only temporarily, as for instance where there is an increase of combustion caused by some exertion. During moments of rest, the means which they possess of conveying without disagreeable exertion a sufficient quantity of oxygen to the blood, and giving off the carbonic acid produced in the body, are amply sufficient.

On the other hand a continuous and considerably increased frequency of respiration, associated with dyspnœa, constitutes one of the most troublesome symptoms of pulmonary phthisis.

Increased frequency of respiration, and the corresponding dyspnœa of phthisical patients, are due partly to a diminished respiratory surface, partly to the accompanying catarrh, which narrows the apertures of the bronchi, partly (though rarely) to the pain caused by respiration, and finally and chiefly to the existing fever.

As a rule, then, dyspnœa is present only where

two or more of these conditions exist at the same time. For instance, the respiratory surface may be exceedingly diminished, without the patient experiencing dyspnœa, or without his respiration, during rest, being accelerated to any great degree, provided at the same time there exist no considerable catarrh, nor pain on respiration, nor fever. Many patients whose lungs have been consolidated and destroyed to such an extent that scarcely one half of the pulmonary capillaries is left to accomplish the interchange of gases, have still the normal number of respirations so long as they are quiet or remain in bed. This phenomenon is easily explained by the fact, that healthy individuals under ordinary circumstances require a very small portion of the whole respiratory means at their disposal to satisfy the respiratory need. Moreover it must be remembered that, in condensations and degenerations of the lung tissue, those alveoli, which have escaped the ravages of disease, admit and expel during respiration more air than the alveoli of an entirely healthy lung. This increased interchange of air compensates to a very great extent for the loss of lung tissue.

A great number of alveoli become blocked up, and numerous capillary bronchi are closed by tubercular granulations, diminishing thus very considerably the extent of respiratory surface. Generally this condition of things is not appreciated by a physical examination. An extreme frequency of respi-

ration, without dullness on percussion, and without bronchial respiration, belongs therefore to the most important symptoms of tubercular phthisis in its restricted sense. If a patient who has condensation and degeneration of a portion of his lungs, and who has previously suffered little or not at all from want of breath, becomes seized with great frequency of respiration and troublesome dyspnœa, without our being able to explain this phenomenon by any physically demonstrated increase of condensation and degeneration of the lung, or by an increase of fever, we must then be suspicious that the existing phthisis has become complicated by tuberculosis. There are cases in which we can conclude that this complication has taken place, by the want of correspondence between the small extent of dullness and the great frequency of respiration.

A short time since, there died at the hospital a stone-cutter, fifty-one years of age, in whom a phthisis of several years' standing, with a moderate amount of condensation and degeneration in both apices, became complicated by tuberculosis. In this case, all the ordinary symptoms which generally render the diagnosis of tuberculosis easy, were wanting, i. e., laryngeal, intestinal, or cerebral phenomena; still, we were able to diagnosticate with certainty during life the commencement of a tuberculosis, from the fact that the patient's respirations numbered habitually from 48 to 54 per minute, with a pulse of 104

to 112, and a temperature varying from 100·5° to 102° Fahrenheit. At the same time there was no appreciable extension of dullness over either apex.

That the frequency of respiration in phthisical patients should be increased by pleuritic pains, by an extension of the bronchial catarrh which accompanies phthisis, by the complication of pleuritic exudations, hydro- and pneumo-thorax, etc., requires no further explanation.

The necessity for respiration augments in fever just as it does in every physical exertion, because in both cases there is an increase as well in the production of carbonic acid, as in the consumption of oxygen. If we compare the frequency of respiration with the temperature and the pulse in phthisical patients, it appears that the increased need of respiration is *partly* satisfied by a greater depth of inspiration, for the evening rise in temperature and pulse does not correspond with the increase in number of respirations; the latter seldom showing an exacerbation of 6 to 8, or even 3 to 4 per minute, above the morning rate. Often there is no increase at all.

Pains in the breast and shoulders are often absent during the whole course of the disease. In general they accompany the pneumonic process, more frequently than the tubercular. (Compare the foregoing case of Dr. N.) In cases, therefore, where the physical examination gives us no clue, and we are doubtful whether we have before us small dispersed

pneumonic masses or tubercles, the pleuritic pains may be of some aid in the diagnosis and prognosis, especially when sputa colored with blood are present.

In numerous cases *cough and expectoration* precede pulmonary phthisis for a longer or shorter period, and belong then to the prodromal catarrh, which eventually leads to pulmonary phthisis, by its extension to the alveoli, and by the caseous degeneration of the inflammatory products there produced.

It is of the greatest importance to ascertain in every case, whether the fever, loss of flesh, paleness, etc., have appeared subsequently to the cough and the moderate expectoration, or whether these symptoms have appeared coincident with the cough and dyspnœa, but yet before the expectoration had become considerable. In the first case, it is quite probable, *cæteris paribus*, that we have to do with pneumonic processes; in the second, however, with tubercular phthisis. Heretofore it would have been said that in the first case the fever and loss of flesh appeared in an advanced stage of tuberculosis* of the lungs, but that in the second case, the various symptoms appeared simultaneously.

The duration of the prodromal catarrh varies. It

* According to Louis, this is the case in four fifths of the patients, while it is only in one fifth that the fever begins at the same time with the cough.

happens sometimes as early as the second or third week that clear signs of an invasion of the alveoli and of a so-called phthisis incipiens show themselves. In this category must be placed those cases in which phthisis has followed directly upon rubeola or pertussis, and especially many of those cases in which phthisis is said to have begun under the form or mask of a catarrhal fever or an influenza. On the other hand, a catarrh may last for months or years, becoming worse in winter, better in summer, until finally it invades the alveoli. In such cases the physician has felt no alarm, because, notwithstanding the cough and expectoration, the patient has had no fever, has preserved his strength, and has remained in a good condition of nutrition. But the scene suddenly changes and the symptoms of phthisis then appear.

Moreover, the original seat of the catarrh which has this serious termination, varies in different cases. Sometimes it is at the onset a catarrh of the finest bronchial branches, but frequently the catarrhal disease begins in the larynx or trachea and extends later to the more dangerous regions, from which there may be a further extension into the alveoli. In my work on pathology I have made a quotation from the "Clinique Medicale"* of Andral, in which it appears that

* Niemeyer, Lehrbuch der Speziellen Pathologie und Therapie. IV Auflage. Bd. I, §. 237.

he had frequently observed such cases as have just been mentioned, but had given them a different interpretation.

A striking example of this form of the disease was offered in the case of a young peasant, who, the day after being mustered into military service, awoke completely aphonic, with a hoarse barking cough; he attributed his sickness—no doubt correctly—to the excess of the previous day, and paid but little attention to it. The aphonia disappeared after a few days, and the cough lost its hoarseness, when a fever set in. The patient now expectorated copious and somewhat bloody sputa, and gradually lost flesh and strength. Two months later, when he presented himself at the hospital, we found dullness over the right apex, extending down as far as the third rib. When mustered into the service his lungs had been found to be perfectly healthy.

We can judge, to a certain extent, of the probability of a catarrh leading to phthisis by the general condition of the patient as exemplified in a previously mentioned case.

In general, persons who have suffered very frequently from catarrh, and whose previous attacks have lasted a long time, are more liable to this danger than those in whom this predisposition does not exist. Still the latter possess no immunity, as is shown by the case we have just mentioned of the

peasant, who was previously a strong and healthy man.

The *expectoration* in the course of an ordinary catarrh is deserving of the greatest attention. We do not regard the fine, sharply limited, saturated, yellow stripes, which are seen in the expectoration,* as a sign of a commencing tuberculosis, but rather of a catarrh of the finest bronchi with copious cellular products; and we know that when the catarrh has this location and form, we have the greatest fear that it may invade the alveoli. Moreover the intimate admixture of blood with the mucus and mucopurulent sputa is a dangerous sign, because as a rule it signifies the beginning of a pneumonic process.

During the disease itself, cough and expectoration are rarely wanting; still I remember the case of a young woman, eighteen years of age, who, although entirely free from cough and expectoration, but with considerable fever, had lost flesh to an extreme degree, and in whom to my astonishment I found the whole upper lobe of the left lung consolidated. Here the pneumonic infiltration had apparently taken place without a previous or contemporary disease of the mucous membrane. It is known that cough and expectoration sometimes almost entirely disappear, as, for example, where an accompanying in-

*Louis, crachats striés de lignes jaunes.

testinal tuberculosis sets up an abundant diarrhœa, a phenomenon probably due to the irritation of the bronchial mucous membrane being lessened by that of the intestine.

A tedious and harassing cough with scanty expectoration, not rich in organized elements, and corresponding to the "sputum crudum" of the ancients, or to the "pure mucous sputa" of more modern authors, is highly suspicious. *Cæteris paribus*, it is a ground for fear that we have to do, not with pneumonic processes, but with a tuberculosis of the bronchial mucous membrane and the alveoli; and we fully agree with the statement of Canstatt "that it is a symptom which excites to the highest degree a suspicion of tuberculosis, when, associated with an obstinate cough and fever, the sputa retain for a long time their rough character, as if from an acute bronchitis."*

The other properties of the cough and expectoration, which are given by authors as characteristic of tuberculosis, we may indeed regard as symptoms of phthisis, but by no means as symptoms of tuberculosis. The sputa globosa fundum petentia indicate globular cavities; the rice-formed corpuscles in the expectoration, if they do not come from the tonsils, indicate a diphtheritic degeneration in the

* Canstatt, Specielle Pathologie und Therapie. Dritte Auflage, von Dr. Henoch. Erlangen 1855. Bd. II.

wall of a bronchus or of a cavity; the sinking of the sputa in water denotes that they have not originated in the large bronchi, where they would ordinarily be mixed with a great quantity of air; the discovery of elastic fibres in the expectoration is a sure sign of the process of destruction, etc.; but we must however seek after other signs of tuberculosis.

For we can boldly assert, that a patient who, in connection with other symptoms of pulmonary phthisis expectorates sputa from whose character we can infer the existence of extensive destruction of the lung, is often in less danger than a patient who is feverish, pale, and thin, and expectorates only tough, transparent sputa. It happens quite often that patients who have lost flesh to an extreme degree, being placed under favorable circumstances, live for a long time, increase in strength, and gain many pounds in weight. My reports of cases contain numerous proofs of the correctness of this assertion.

A hoarse or *toneless cough* belongs to the most important symptoms of *tubercular phthisis*, or of the *complication by tuberculosis of a pulmonary phthisis originally depending on destructive inflammatory processes.* The highly interesting cases of phthisical patients, in whom the alteration of the voice and the tone of the cough is the result of paralysis of the vocal chords caused by pressure upon the recurrent laryngeal nerve by pleuritic forma-

tions, can scarcely be compared in frequency with the cases in which these symptoms are caused by tuberculous ulceration of the mucous membrane. If the cough does not become rough and toneless until an advanced stage of the phthisis, we are to suspect a consecutive tuberculosis. If it has had that character from the very beginning, and especially at a time when the sputa are still tough and transparent, and the physical investigation of the chest discloses no considerable alterations, we can then very reasonably entertain the suspicion of a primary tubercular phthisis. It is not rare, as we have previously stated, for a tuberculosis to begin in the trachea and larynx and extend later into the finer bronchi.

Fever is one of the constant symptoms, as well of chronic pneumonia as of tuberculosis. Ziemssen has shown that in children the invasion of the alveoli by the catarrh is invariably accompanied by a considerable increase in the temperature and pulse. This is equally true of the beginning of a catarrhal pneumonia in adults. The statement of Louis, that in the majority of patients (four fifths) the fever does not become associated with tuberculosis until at a more or less advanced stage, is explained by the fact, that that investigator viewed things from Laennec's standpoint, and therefore deduced the prodromal catarrh from an already existing tuberculosis. We have repeatedly called attention to

the dangerous results of this error, and believe we can positively affirm that by a strict attention to the temperature and pulse in every simple catarrh, and by a most careful treatment of every case of prolonged catarrh, in which fever begins to show itself, the development and extension of pulmonary phthisis can very often be warded off.

Just as the appearance of the fever is an important sign of the extension of a catarrh from the bronchial mucous membrane to the alveoli, so is its continuance the most important proof that the pneumonic processes have not reached their termination. The curves formed by the graphical representation of the morning and evening temperature of phthisical patients, show ordinarily a striking sameness, and we can deduce from them the presence of pulmonary phthisis with almost the same certainty as is the case with typhoid fever or acute croupal pneumonia. The difference of the morning and evening temperature is as a rule from 2° to 3°, rarely smaller, and very often greater; we find often that the temperature in the morning is almost normal, while in the afternoon and evening it ascends to 102° or higher. This course is not indeed peculiar to all consumptive fevers. Upon comparison of the temperature curve of phthisis with that of tedious suppurations at the periphery, caries, etc., we find a great difference, especially concerning the regularity of the morning remissions and the evening

exacerbations. We have not entirely finished our investigations concerning the hectic fever of phthisical patients, especially in regard to the influences by which the regular course of the same is interrupted, still we can even now affirm, that in *tubercular phthisis* in its restricted sense, and in a tuberculosis which has appeared secondarily in the course of a destructive pneumonia, the variations between the morning and evening temperature are as a rule much less considerable. Therefore, *cæteris paribus*, as long as the fever is a febris remittens with an almost intermittent form, our prognosis is better than when the fever is a continuous one. In phthisis we have succeeded in numerous cases in moderating or entirely dispelling the fever, and thereby improving considerably the patient's strength and condition of nutrition; while in patients in whom no morning remissions occurred we can show no such results.

If the caseous masses become encapsuled or subsequently liquefied and reabsorbed, the fever may entirely cease, and it is not rare to see patients, with extensive cavities in the apices of the lungs, entirely free from fever.

In those cases in which the pneumonia has terminated in induration, the physical signs and the globular sputa expectorated day by day, especially in the morning, contrast in a striking manner with the subjective condition of the patient, with his fresh and healthy look, and with the condition of

his forces and nutrition. We have sufficiently demonstrated that such persons, notwithstanding the termination of their previous illness in relative recovery, are nevertheless in danger, both from a relapse of the pneumonic processes, and from a phthisis resulting from a consecutive tuberculosis; and we must recommend as very practical in such cases to continue once in a while the investigation of the weight and temperature, in order to assure ourselves whether any of the foregoing events have occurred.

From the preceding, it is evident, that thermometry has in pulmonary phthisis a great or still greater importance for the diagnosis and for the prognosis, as well as for the treatment, than in any other disease.

The impoverishment of the blood and the loss of flesh, symptoms from which arises the term "consumption," are mentioned by us after the fever, because we have no doubt but that the fever is the principal source of the same. The most convincing proof of the correctness of the theory that the increase of temperature is due to an increased combustion of the tissues, is the rapid decrease in weight even in a fever of brief duration. It has been proved in my clinic for a series of years past, by numerous investigations* of temperature and

* Compare the publications of Liebermeister in the Prager Vierteljahrschrift and in the Deutsches Archiv für Klinische Medicin.

weight, in patients with pulmonary phthisis, that the decrease and increase of weight correspond with the presence or the absence of the fever.

The belief that a continued fever of ordinary intensity, especially when the patient continues in bed, consumes less rapidly than a fever in which, as in the hectic fever of phthisical patients, the temperature ascends daily from about the normal point to a considerable height, is very enticing. At any rate the production of heat and the combustion of the elements of the body is, as is shown by Zimmermann,* during the rapid increase of temperature, very considerable, but we nevertheless hesitate to acknowledge the correctness of the hypothesis. The recognition that it is especially by the fever that the forces and flesh of phthisical patients are consumed, is of the greatest importance in the treatment of pulmonary phthisis.

Again, the *physical signs*, which have h eretofore been considered as characteristic of pulmonary tuberculosis, acquire a different signification when we depart from Laennec's views, and consider it necessary in every case to ask ourselves whether we have to do with a tuberculosis, or with a disintegration of caseous infiltration, or with inflammatory processes, which have led to a wasting and shrinking of the pulmonary parenchyma.

* De Morbis Febrilibus Quæstiones Nonnulæ. Inaugural Dissertation. Berlin. 1860.

Although these different conditions are often combined with each other, still it can *by no means* be claimed that this is always the case; and even when such a combination exists, it is of importance to ascertain what form of nutritive disturbance prevails. We will therefore subject to a brief examination the physical diagnosis of pulmonary phthisis as regarded from our standpoint.

It is quite possible for an individual with a *paralytic thorax* and *phthisical "habitus"* to possess perfectly healthy lungs, but in such a case we assume, as we have previously explained, the existence of a certain weakness or vulnerability that stands in close relation to protracted catarrhs, and such other diseases as leave behind caseous products. The existence of this "habitus" can therefore be of some importance in the diagnosis of doubtful cases.

Since, however, a predisposition to these diseases includes to a certain extent a predisposition to tuberculosis, we must, in the case of an individual with a paralytic thorax, especially if some consolidation be discovered at one or other apex, consider very carefully the possibility of there having been developed a consecutive tuberculosis.

It is generally acknowledged that the physical signs "*in the first stage of pulmonary phthisis*" depend only on the catarrhal swelling of the bronchial mucous membrane, and the secretion in the bronchi. According to the prevailing views the

tubercles and tubercular conglomerations have not yet coalesced to form extensive condensations at the periphery, so long as the only physical signs are rude and diminished vesicular respiration, interrupted respiration, prolonged and harsh respiratory sound, ronchi, and mucous râles over the apex. But, "since a catarrh, limited to the apex of the lung, is always due to an irritation exercised by tubercles on the adjacent tissue, it is a *certain sign of a commencing pulmonary tuberculosis.*" This opinion stands in direct opposition to the results of pathologico-anatomical investigations.

At autopsies we find in the apices of the lungs pneumonic processes of more or less recent date, induration, shrinking of the pulmonary tissue, and cavities, which are of bronchiectatic origin, or due to a disintegration of caseous infiltration, much more frequently than we find tubercles. It is very evident that catarrhs affecting the apex of the lung have a special tendency to invade the walls of the bronchi and their lateral and terminal alveoli. So long as the peribronchitic and pneumonic deposits, which have thus originated, do not coalesce to form extensive condensations, dullness and bronchial breathing are of course absent, and the physical examination gives, with the exception of the signs of catarrh, a negative result. Hence we regard a protracted catarrh at the apex as a dangerous circumstance. The longer it is prolonged, so much

more are we justified in the fear that it will lead, or has already led, to those morbid conditions of nutrition which cause most frequently pulmonary phthisis. We conclude that the catarrh has really invaded the bronchial walls and pulmonary parenchyma, principally, as has previously been explained, from the appearance of fever, the disturbance of the general health, loss of flesh, and paleness. But even in such cases we have, by careful *treatment*, so frequently seen complete recovery that we cannot acknowledge that the sole termination of the pneumonic processes is in caseous infiltration and disintegration, that is, in pulmonary phthisis. Nor can we regard a catarrh affecting the apex as a sure sign of a commencing pulmonary tuberculosis, nor as a constant forerunner of pulmonary phthisis, but indeed as a *sign* that the patient is in *danger of becoming phthisical.*

Dullness on percussion, bronchial respiration, and *consonating mucous râles in the upper portions of the chest*, are regarded as pathognostic of pulmonary tuberculosis or phthisis.

Most patients know, when they consult a new physician, how large the extent of the dullness was at the last examination. Since dullness on percussion and bronchial respiration never originate in the coalescing of tubercles and tubercular conglomerations, we can conclude with certainty from those signs, the existence of a pneumonic infiltration or

its results. By this we do not mean to say, that a patient with dullness and bronchial respiration in the apex cannot also have tubercles. On the contrary, on returning to the symptoms, we are permitted to conclude that in connection with the results of inflammatory processes in the lungs, tubercles are sometimes present; but in general, the following statement can be considered as true: if the extension of the dullness and bronchial respiration stand in direct proportion to the advance made by the phthisis, the prognosis is more favorable than in a case where the patient rapidly loses flesh without the extension of the dullness keeping pace with the advances made by the other morbid phenomena.

In the former case, we have to do principally with pneumonic processes, in the latter, probably with a tubercular deposit.

It is not dullness, but a dull tympanitic sound on percussion met with in a patient with great frequency of respiration, cough, scanty expectoration, etc., that must awaken suspicion that the æration is diminished, and the pulmonary tissue impaired by a tuberculosis.

A short time since, a patient was received into the hospital, with serious cerebral symptoms. The dull tympanitic sound on percussion, the diminished depth of respiration, and the weak vesicular breathing, aided us materially in making, in this case, the

especially difficult diagnosis of a tubercular meningitis; we concluded, principally from the physical signs just mentioned, that very probably there existed an extensive miliary tuberculosis of the right lung, and we gained thereby an important point for the interpretation of the cerebral symptoms, the correctness of which was confirmed by the autopsy. We must add here, that tympanitic dullness on percussion may also be due to scattered pneumonic infiltrations.

The sinking in of the supra- and infra-clavicular regions on one or both sides, which has hitherto played so great a part in the symptomatology of pulmonary tuberculosis, as well as the *lowered situation of the upper limb of the lung*, signify neither a tuberculosis, nor a caseous infiltration, nor a disintegration of the pulmonary parenchyma, but always and only a diminution of the apex of the lung from induration and contraction. The same process which has caused the sinking in of the thorax, can, indeed, lead at the same time to the formation of bronchiectatic cavities, and the walls of these can become the seat of a diphtheritic destruction. Moreover, at other points of the lungs, caseous infiltrations can be undergoing the process of disintegration, or tubercles can be dispersed throughout the pulmonary parenchyma, but we must have for the diagnosis other symptoms in the general condition of the patient, in the expectorated sputa, or in other

physical signs, before we can declare that an individual with sunken supra- and infra-clavicular regions is phthisical. If we have none of these corroborating points, then the symptom referred to is to be considered as an important sign of a favorable termination of a process which is so frequently the foundation of phthisis. Such patients have, of course, no immunity against phthisis, but are, on the contrary, as we have already sufficiently demonstrated, in much greater danger of becoming phthisical from fresh pneumonic attacks, or a consecutive tuberculosis, than individuals without contraction and induration of the apices of the lungs.

The diminished respiratory movement of the superior portions of the thorax has, when it corresponds with the regions that are sunken in, the same signification. It may happen in such cases, that the shrunken parenchyma, which has also become impermeable to air, cannot yield to the force exercised by the muscles of inspiration, and that these points of the thorax may thus retain their form; but if there is dullness above these points, we can conclude that there is an extensive condensation of the parenchyma, most probably due to a pneumonic infiltration. Diminished respiratory movement, at a point where percussion discloses no dullness, though perhaps a somewhat dull and tympanitic normal sound, excites suspicion of tubercles, but it is no sure sign, since small scattered pneumonic masses

can diminish the respiratory movement without causing any dullness.

Finally, the *cavernous sounds*, including the tolerably frequent metamorphosing (Seitz) respiratory sounds, probably do not often occur in tuberculous cavities. I consider it on the whole questionable whether extensive cavities originate in the softening and emptying out of tubercular conglomerations, and also whether this same process is repeated in the walls of a primary cavity by a subsequent tubercular development. At any rate, most of the large cavities in phthisical lungs have mainly a bronchiectatic origin, or have originated in the disintegration of a caseous infiltrated tissue. The well known combination of symptoms, which is considered as pathognomonic of bronchiectatic cavities, holds good only in bronchiectases in the inferior lobes of the lungs, where the contents of the cavities are thrown out with difficulty, and therefore easily undergo decomposition. The contents of bronchiectatic caverns in the apices, which are very much more frequent than those of the inferior lobes, can easily be emptied out, so that it is very seldom that decomposition occurs.

If we hear cavernous sounds in the apex of the lung, without the supra- and infra-clavicular region being sunken in, and without the upper border of the lung being lower than normal, we can conclude, with a certain amount of probability, that

the cavity has originated in the disintegration of the pulmonary tissue; on the contrary, if there exists in connection with cavernous sounds the signs of induration and contraction of the apex, we can infer that we have to deal with a bronchiectatic cavity. It is by no means rare, that persons with the last mentioned symptoms continue for a long time to feel comparatively well, retain their full strength, and rather increase than decrease in weight until they are seized with a new pneumonic attack or a consecutive tuberculosis.

In conclusion, we will endeavor to disclose the principal features under which the different clinical forms of pulmonary phthisis usually appear, and will in the next place occupy ourselves with that form in which the morbid phenomena are *caused entirely by pneumonic processes and their terminations.*

Phthisis often begins more or less suddenly under the form of an acute disease. Here belong those cases in which a croupal pneumonia, instead of terminating in resolution, leads to a caseous infiltration; also those cases in which the blood from an haemoptysis, having been poured into the bronchi and alveoli, and there becoming coagulated, induces intense and widespread pneumonic processes; and finally, those cases, in which an acute bronchial catarrh invades the alveoli of a considerable portion of the lung.

The rare termination of a typical pneumonia in caseous infiltration and phthisis is to be feared, when at the end of the first or at the beginning of the second week of the disease the fever does not cease when there is a considerable evening exacerbation and it yields during a remission of the fever with copious perspiration, when dullness on percussion continues, and over the same extent there are for some time crepitating mucous râles, and when a considerable quantity of muco-purulent sputa is expectorated.

In addition to these symptoms, the discovery of elastic fibres in the expectoration, and the existence of cavernous sounds, leave no room for doubt that the caseous infiltration is undergoing the process of disintegration. Most patients consumed by the violent fever, succumb in a few weeks. It is of much rarer occurrence, that at a time when the prognosis appears most unfavorable, improvement should take place, the expectoration becoming scantier, and the patients beginning to recover slowly: the dullness, however, continues; the thorax in the same region gradually sinks in: and after a time, pronounced signs of induration and contraction of the diseased portion of the lungs appear, and often also of bronchiectatic cavities.

Very much the same course is pursued in part by those pneumonic processes which follow immediately upon an hæmoptysis or pneumorrhagia, and

which, according to our view, are induced by the coagulation of the blood within the bronchi and the alveoli. The more extensive the dullness is which appears in the course of an hæmorrhage, and the longer it continues, the more pronounced the pleuritic symptoms, the more violent the fever, and the longer it exists, so much the more reason have we to fear that the retained blood and the inflamed pulmonary parenchyma have undergone a caseous metamorphosis, and that extensive destruction of the lung will follow. But the clinical cases which have heretofore been described, prove that even in such case the caseous masses of the lung may, at a later period, become liquefied and reabsorbed, or capsulated, or the diseased portion of the lung may become indurated and contracted by the active proliferation of connective tissue.

However, the invasion of numerous alveoli by an acute catarrh is sometimes accompanied by such serious symptoms, especially by so violent a fever and such a rapid loss of strength and deterioration of nutrition, that the diagnosis may at first be difficult. It is in such cases pardonable if we presume for a time, especially until we find more certain signs, that the catarrh and the violent fever are the result of an infection, or acute tubercular development in the lungs. But the case soon becomes more evident. Some of the sputa show the pathognomonic color of pneumonic sputa, (due to admix-

ture of blood;) more or less violent, and more or less extensive pleuritic pains begin to be experienced; percussion over the upper portions of the thorax shows tympanitic dullness, and if the originally lobular masses coalesce to form extensive condensations, there will be positive dullness; under these conditions the previously indefinite mucous râles become consonating, the respiratory sound bronchial, etc.

These catarrhal infiltrations of an acute origin sometimes undergo complete resolution, but it happens much more frequently that the infiltrated tissue undergoes a caseous metamorphosis, and becomes in a short time disintegrated. Most cases of galloping phthisis, in which, within a few weeks, extensive destruction of the lungs occurs, and the patients lose flesh rapidly, with violent fever, and succumb, originate in the invasion of the alveoli of large portions of the lung by an acute catarrh, and can be designated as the termination of an acute or subacute catarrhal pneumonia in "phthisis florida." If the process in question has extended over an entire lobe, it is rare for subsequent reabsorption or capsulation of the caseous mass, or induration and contraction of the diseased portion of the lung, to take place.

Still, we have had an opportunity to observe for several years at the hospital an exquisite example of this course of the disease in the case of a man

twenty-four years of age. In this person the entire left superior lobe is considerably retracted, the heart lies adjacent to the wall of the thorax for a great extent, the point of the heart is dislocated towards the left, the right lung is the seat of an extensive vicarious emphysema, and projects two finger-breadths beyond the left border of the sternum. If the acute catarrhal pneumonia leaves behind a caseous infiltration of small extent, the termination in induration and contraction is much more frequent.

The depression of the supra- and infra-clavicular regions, and the low situation of the superior borders of the lungs, can be referred in the case of many persons to an attack of acute catarrhal pneumonia, which has been prolonged and has taken the termination in question.

There is frequently offered an opportunity of observing patients who, after longer or shorter intervals, are again and again seized with such attacks, go through with the same fortunately, but derive from every attack an increase in the extent of the dullness and of the depressed regions of the chest, until they are finally carried off by a pneumonic process of less favorable course, or by a tuberculosis.

Contrary to the previously mentioned cases, the extension of a catarrh from the bronchi to the alveoli may occur without sudden and violent symptoms, and even in an entirely latent manner. There are very

often found at autopsies, in the apices of the lungs, cicatrized contractions, capsulated caseous masses, and callous condensations, the residua of pneumonic processes which had entirely escaped observation during life. In the same manner, we find in many individuals the supra- and infra-clavicular regions sunken in, and the upper border of the lung lower than it should be, without our being able to discover at what time, and under what symptoms the pneumonia, which led to induration and contraction of the apex, existed. However, in case of any considerable extension of the process, the chronic catarrhal pneumonia seems also to be constantly associated with fever.

This "slow" fever is indeed, as a rule, overlooked or misinterpreted by the patients, and sometimes also for a *while* by the physician, inasmuch as the more evident subjective febrile symptoms, such as slight chills, feeling of heat, increased thirst, etc., are inconsiderable, and pass unnoticed in comparison with the results of the increased combustion of the elements of the body, the loss of appetite, indigestion, imperfect assimilation, and anæmia.

When a patient with chronic bronchial catarrh, by which his general health and strength has not been diminished, loses appetite, becomes pale and thin, and remarks in himself an evident decrease in strength, the suspicion should be entertained that the catarrh has invaded the alveoli, and it is ear-

nestly to be recommended that careful observations regarding the temperature should be taken, and repeated physical examinations made, to ascertain whether condensations exist in the lungs.

The chronic form of catarrhal pneumonia shows a decided tendency, under favorable external influences, to terminate in induration and contraction, but also to relapse under unfavorable influences. Thus it is explained how innumerable patients, notwithstanding extensive callous condensations and bronchiectatic cavities in the apices of the lungs, are in quite a comfortable condition during the summer months, and increase in strength and weight, while during winter, especially when they must work, and be exposed to "taking cold," they become feverish, weak, pale and thin, and acquire new condensations of the lung. This change is often repeated for a series of years. Such patients form a large quota for the hospitals, and are generally there unwelcome guests, unless they offer some especially rare physical symptoms, inasmuch as "chronic tuberculosis of the lungs" (!) is considered generally as an uninteresting disease.

The great influence which therapeutic, and especially dietetic means in their full sense, exert against this, by far the most frequent form of phthisis, is, when viewed from our position, easily understood, and forms, as it were, a proof of the correctness of these views.

We must continually keep in mind the possibility, *that a tuberculosis may be developed in the course of a pulmonary phthisis caused by pneumonic processes.* We will repeat the sentence which we have previously quoted, "that the greatest danger for most phthisical persons is that they may become tuberculous." This possibility can be realized in every stage, and we must not, even in the most favorable course of a pulmonary phthisis, become too confident or make an unconditionally good prognosis.

The development of tubercles in phthisical lungs can take place in so latent a manner, that the diagnosis can either not be made, or at best not with certainty. In many other cases, however, especially in those in which the lungs are the seat of numerous tubercles, and in which the tuberculosis extends also to other organs, there is no difficulty in the diagnosis. If we find that a patient suffering from pulmonary phthisis becomes very short-winded, without an extension of the dullness over the chest, if the fever continues in spite of the most careful treatment, and if the febris remittens becomes a febris continua, if diarrhœa takes the place of a tendency to constipation which has probably hitherto existed, if there is added to the other symptoms hoarseness and aphonia, or the familiar symptoms of disease of the meninges of the base of the brain, then we can assume, with full confidence,

that in the case before us a tuberculosis has become associated with the phthisis. In young individuals in whom tuberculosis has a special tendency to attack the cerebral membranes, the cerebral symptoms offer the principal diagnostic points, while in older persons the morbid symptoms are referable to the intestines and the larynx.

Finally, the form under which primary tubercular phthisis begins and runs its course, differs materially from the forms hitherto described.

In the first place, the prodromal catarrh is absent. The fever and the "wasting away" is not deferred until the patients expectorate copious muco-purulent sputa; but, on the contrary, the tubercular development, especially when it is extensive, begins with a considerable increase in temperature and a rapid consumption of the body from the fever. If we learn from a patient that he first began to cough and expectorate, after having for several weeks previous rapidly grown weak, pale and thin, we must fear that he is suffering from a tubercular phthisis. This suspicion must be still stronger, if the patient has extraordinary dyspnœa, and if the physical examination of the chest gives at the commencement negative results; at a later stage the sound on percussion may become dull on account of consecutive pneumonic processes, and there may be bronchial respiration and consonating mucous râles, but it is only in certain individual cases that the condensa-

tions of the lungs become as extensive as in the previously mentioned forms of pulmonary phthisis. At an early date, the tone of the voice and cough becomes hoarse, and if the tuberculous disease of the larynx becomes considerable and rapidly spreads, the well known tormenting symptoms of laryngeal phthisis show themselves. Moreover, we do not as a rule have to wait very long for the symptoms of the intestinal tuberculosis and the consequent intestinal tubercular phthisis. The consumption is hastened by the copious diarrhœa, the abdomen becomes tender on pressure, etc. The disease lasts seldom longer than several months, and most patients yield to it still earlier.

It has not been our intention to describe the course of the preceding forms of pulmonary phthisis in a thoroughly exhaustive manner, and to speak of the innumerable modifications which may be due to individual peculiarities, nor to allude to the numerous changes affected by acute and chronic processes, to the various accidents which may ensue, or to the complications and other circumstances. Still, most cases of pulmonary phthisis, which one has himself observed, or taken from good and complete clinical reports, can be without difficulty ranged under one or the other category established by us.

The *treatment* of pulmonary phthisis has gained a much firmer foundation on account of the recognition of the fact, that we are in this disease concerned, as a rule, with pneumonic processes, and only in isolated cases with a morbid deposit. We have not indeed found any new or positive "cures for phthisis," but we have, it is true, by our investigations been placed in a condition to establish clearer indications for the use of our ordinary remedies, and in many cases to obtain, by the constant application of these means, better results than were attained at the time when pulmonary phthisis was considered just as incurable as a cancerous disease, and treated accordingly.*

In respect to the prevention of pulmonary phthisis; in the first place, in regard to individuals of a weak constitution and in whom there are already present evident signs of a great vulnerability and a disposition to diseases resulting in a formation of caseous products, it is our duty to place them, when

* Most physicians, in regard to the curability of phthisis, still adhere, even to-day, to Laennec's views: "Presque tous les hommes de l'art, qui sont au courant des progrès récens de l'anatomie pathologique pensent au contraire aujourd'hui que l'affection tuberculeuse est, comme les affections cancéreuses, *absolument incurable* parceque la nature ne fait que des efforts contraires à la guérison et que l'art n'en peut faire que d'inutiles." (Laennec, ii, 58.)

it is possible, under such influences as will favor a strengthening of the constitution and the rooting out of those morbid dispositions.

Without going particularly into the dietetic rules indicated under such circumstances, I shall only call attention to a very frequent and palpable error in this respect. Careful mothers, especially those who have already lost children from croup, capillary bronchitis, etc., and who, on account of the repeated sickness of the remaining children, are very careful of them, are accustomed, in order to protect them from taking cold, and often with the advice of the physician, to diminish their stay in the *open air*. The injurious influence which a constant confinement to the house has upon the organism, is, indeed, not yet sufficiently explained: but it is fully proven that scrofula, as well as pulmonary phthisis, occurs much more frequently in foundling and orphan asylums, houses of correction, and prisons, and among those employed in factories, who are occupied the whole day in close apartments, than among persons who move about a great deal in the open air. The argument that the frequency of scrofula and pulmonary phthisis in such institutions is caused by other influences than the absence of fresh air, and especially by bad and insufficient food, cannot be demonstrated. The inhabitants of many poor villages live, on an average, on much worse food, and are exposed to more numerous injurious influences,

than the persons detained in prisons and houses of correction, and still they are not equally subject to those diseases. *Too little attention has hitherto been paid to these striking facts in practice.* Numerous weak and miserable children, with protracted catarrhs, chronic exanthemata, swollen lymphatic glands, etc., are sent yearly to take a course of brine baths, but are allowed during the remaining portion of the year, if they only take their cod liver oil regularly, to sit upon the school bench six hours daily, to prepare their school exercises at home, to have private lessons, to practise on the piano, etc., as if, indeed, cod liver oil could take the place of fresh air. As long as this error prevails among physicians, it is not surprising that many anxious mothers retain their children in-doors during the extent of the winter. Only in cases where the disadvantageous results of this proceeding are especially evident, as (on account of the length of the winter) for example in Russia, it is almost generally the custom to send to the south during winter, not only patients who are already phthisical, but also persons in whom it is feared there appears a disposition to phthisis. It would be desirable that a similar practice should also be introduced among us; that the attendance at school be immediately limited, as soon as the injurious influence begins to be felt, and that when circumstances allow it, weak and sickly children, showing a tendency to catch cold, and scrofulous

children, should be sent during the northern winters to regions where they can daily remain in the open air.

I have met the most stubborn opposition to such, in this part of the country, almost unheard-of advice, but I have in a few cases, where I succeeded in entirely putting a stop to study, and allowing the children to spend the greater portion of the day in the open air, obtained results which surprised myself, and convinced the parents that the results richly compensated for the sacrifice made by them.

As soon as we have freed ourselves from the doctrines of Laennec and certain authorities of modern times, and believe that protacted catarrhs often lead to phthisis, we shall at once admit the necessity of rules for the prophylaxis of pulmonary phthisis, by which, especially in presence of a disposition to phthisis, bronchial catarrhs may, if possible, be avoided, and by which even light bronchial catarrhs may be rapidly and completely cured.

If an acute catarrh invades the alveoli from the bronchi, it is, as far as the course and termination of the disease are concerned, often of decided influence whether the physician considers the symptoms in the apices of the lungs the fever, the serious constitutional disturbance, the rapid loss of strength, etc., as sure signs of a tuberculosis, or whether he assumes that this complex of symptoms can depend

also upon a genuine catarrh and upon lobular pneumonic processes.

In the first case, energetic treatment is considered as a rule useless, and is not advised. The patient is allowed to attend to his business until the increasing fever or the weakness, gaining the upper hand, make it necessary to remain at home, or until bloody sputa, pleuritic pains, the appearance of dullness on percussion, demand that the supposed "secondary" pneumonic and pleuritic processes be subjected at least to a symptomatic treatment. Let no one say that this is an exaggeration; this *laisser aller* is an every-day occurrence in presence of the conditions in question. It is rare that a physician censures himself as being the cause, by his own carelessness, of the development of a phthisis florida, because the conviction is so firmly established that even in this form of pulmonary phthisis we are concerned with the infiltration of the lungs with a morbid deposit, against which therapeutical agents are completely powerless.

Even in case the physician correctly recognizes the nature of the disease, he may not, indeed, be able to cause an immediate abortion of the same, but he has every inducement to advise the most energetic action: he will avail himself of every means to preserve the patient from injurious influences, and will place him under conditions from which it can be expected that the extension of the pneumonic pro-

cesses will be arrested, and its termination in caseous infiltration and disintegration of tissue prevented.

The results of such a proceeding confirm in a striking manner the correctness of the investigations of Virchow, Maier,* Colberg,† and others. To these and to the present position of pathological anatomy is due all the credit.

Let the trial be made of confining for a time strictly to bed patients with so-called signs of a beginning pulmonary tuberculosis. Forbid them all unnecessary speaking and coughing; cover the breast with cataplasms, and at the first appearance and every repetition of pleuritic pains, prescribe a local abstraction of blood by leeches or cups, and we will soon be convinced that many patients become healthy, concerning whom it had hitherto been supposed that they were suffering from tuberculosis, and that there was no help for them. I am, from the experience which I have had of late years, perfectly convinced that I have lost in earlier times many patients from galloping phthisis only because I had considered them from the beginning as lost, and had not treated them as patients should be

* Mittheilungen aus dem Pathologischen Institute in Freiburg, von Rud. Maier.

† Im IV. Hefte des zweiten Bandes vom Deutschen Archiv für Klinische Medicin, von Ziemssen und Zenker. Appeared while the original of these lectures was in press.

treated who are suffering from a pneumonic process, but like patients in whose lungs a morbid formation has been developed, to retard the extension, and the latter metamorphosis of which does not lie in the sphere of medical aid.

This course of action is most earnestly to be recommended also in *the exacerbations which occur in the course of a chronic pulmonary phthisis under more violent febrile symptoms.*

How great is the influence of a treatment that recognizes an inflammatory process in the lung, can be shown by numerous clinical reports.

In the Tübingen clinic, which is principally recruited from among the inhabitants of the neighboring regions, it is not rare to see phthisical patients during a period of several years admitted from time to time and treated. They generally spend a few weeks in the hospital, and during that time are treated in the aforesaid manner.

The fever, the weakness, the loss of flesh, which have often reached such an extreme degree on their admission that the very worst was feared, disappear, and the patients themselves demand dismissal, in order to return to their occupations. It is of course often but a short time before they return with violent fever, extreme weakness, and a loss in weight of eight or ten pounds, and beg for readmission. But not only my experience in hospitals, but also that in private practice, has convinced me

how great an advantage it is to regard the "hectic" fever of phthisical persons, as a symptom of a chronic inflammation of the lungs, and to make use of the elevation of that fever in the same manner as we do in the fever of an acute croupal inflammation of the lungs, as an index of the intensity, and respectively of the progress of the invasion of the pneumonic process. It is daily the case that phthisical persons, *even when they have at evening tolerably high fever*, are not earnestly enough urged by their physicians to withdraw themselves from their occupations, to cease their work in the counting room or office, and to remain away from clubs and coffee houses filled with tobacco smoke. It happens often enough that these very injurious influences, to which the patients expose themselves on such occasions, assist materially the invasion of the chronic inflammatory processes, and the termination of the same in disintegration, while by a careful avoidance of these influences, by perfect quiet, uniform temperature, by the application of moist heat, etc., it is not rare to see a marked and unexpected check of the disease, followed by great improvement. I have observed in Grieswald the following highly interesting case.

Mr. V., a well-to-do carpenter and good workman, but at the same time given to the enjoyment of a good table and "jolly companions," had suffered from a chronic pulmonary phthisis of several years'

standing, having large cavities in the apices of his lungs. He felt generally comfortable enough during the summer, and went regularly through the cure at some of the springs, most frequently at Ems. During the winter he was in a worse condition, and complained considerably; he still, however, continued at his usual work, and frequented the tavern, according to his daily custom. In the winter of 1858 I lost sight of him, as he had passed into other hands. One day, however, he sent for me, and I was struck with his appearance. He was extremely emaciated, and related that he had lost about fifteen pounds since autumn. The frequency of the pulse was, at evening, 110 to 120, temperature about 102° F., (39° C.) But even in this condition he had been daily at his shop, and had visited the tavern as of old. Judging from the numerous prescriptions that he had, during the past few months, taken great quantities of medicine, I ordered in the first place to discontinue them all, and that the patient should remain in bed for a week, talk as little as possible, suppress the desire to cough, and cover his breast with cataplasms. Herr V. was in a doleful frame of mind on hearing this advice. He pitifully declared he would never be able to leave his bed again, and would soon fall a prey to his disease. But at the end of *one* week the fever had diminished considerably, his night sweats, previously quite copious, had ceased, and his appetite returned.

Mr. V. recovered quite rapidly, and lived still three years after that time.

If a moderate diaphoresis, the application of moist heat to the chest, and the warding off of all influences which may be injurious to the inflamed lung do not have the desired result, and if the fever continues to any degree, we must then avail ourselves of the application of *antipyretic means*. These have on the chronic pneumonia of phthisical persons just as little direct influence as on acute croupal pneumonia, typhoid fever, and other diseases, for which they are frequently prescribed, and often, of course, without a clear understanding of what they are intended to accomplish. If we recognize that the loss in mucous and cellular elements, (which in simple bronchial catarrh is often much more copious,) plays only a small part in the wasting away of phthisical patients, but that the fever is the chief wasting element, then it is self-evident that we must use every means in our power to combat the fever. Among those which are in many cases successful in checking the increase in the production of heat, notwithstanding the continuation of the disease, digitalis and quinine stand justly in the highest repute.

We administer these to phthisical patients very frequently, when we have not succeeded in the aforesaid manner in becoming master of the fever; and pills, containing 1 gr. quiniæ sulphatis, ½ gr.

digitalis, and ½ gr. opii, to be taken four times daily, are most frequently prescribed. At the hospital, the pills are suspended as soon as an evident decrease in temperature and frequency of pulse takes place, and we return to them again as soon as this influence has disappeared. In private practice, I have often noticed that patients learn very soon from their own experience to judge when it is time to suspend the pills, or when the time to resume them has returned.

Having considered the antipyretic treatment of pulmonary phthisis, we will next take into consideration the dietetic rules to be recommended in this disease; and we follow this order for the same reason that, in enumerating the symptoms, we first considered the fever and afterward the wasting away.

A man who has a fever, and whose body is thereby rapidly being consumed, has a much more pressing need of new nutritive material than a man devoid of fever. A patient suffering from pulmonary phthisis has often a fever lasting for months, so that the danger to him of really being consumed by it, is much greater than to a man who is suffering for a short time from an acute febrile disease. It is therefore evident that we have a special inducement to supply phthisical patients with nourishment as rich and as suitable as possible.

It is often asserted that the nourishment increases

the fever: but nothing is further from being proved; and here among us, not to speak of the practice in England, patients are put upon the so-called fever or low diet, and kept on it until it appears dangerous to continue it any longer.

When the latter is the case, this empirical law is entirely ignored, or rather, an opposite treatment is instituted. In the choice of nourishment suitable for phthisical patients, we find that the old articles chosen empirically agree with those established by laws of physiology. All of the articles of food principally recommended to phthisical patients contain great quantities of fat substances forming fat, and comparatively few protein substances. The experimental fact that consumption of protein substances increases the production of urea and the metamorphosis of the nitrogenous elements, but that on the contrary, by the consumption of the fats, etc., the metamorphosis and combustion of the organs and tissues, which are the most important for the organism, are decreased, corresponds to this choice of food.

We cannot then recomend too earnestly to phthisical patients as copious a use as possible of milk (to which indeed children owe the round form of their limbs, and which corpulent individuals avoid with good reason.) But it is entirely useless and unreasonable to extract the casein from the milk nd allow only the whey, unless indeed the patient,

which is extremely rare, can easily digest the whey but not the milk. When I order for my patients a pint of milk three times daily, *fresh from the cow*, I have no other reason for so doing, but the prevention of its being deprived of cream, which is of course impossible immediately after the milking. I prize cod liver oil very highly, but it can very well be replaced by the extract of malt, which is less nauseating and lighter for the stomach, and which, instead of the fat, contains substances which form fat, in a form easily to be assimilated.

Moreover the grape cures are recommended as replacing the cod liver oil.

Strange to say, they are considered by many physicians as apt to cause diarrhœa, and are therefore sometimes prescribed as an after-cure, after a season at Marienbad or Carlsbad. Under a daily consumption of three to four pounds of sweet grapes in Vevey, Montreux, etc., almost every one becomes in a few weeks fatter and heavier than they were before the cure, and if the intestines were previously healthy it is seldom the case that diarrhœa occurs.

As for the rest, pulmonary phthisis, depending upon pneumonic processes, must be treated *according to the same principles, which prevail in the treatment of catarrhs limited to the bronchial mucous membrane.* It would lead us too far to define more exactly under what conditions the administration

of the so-called expectorants is indicated, or what conditions are suitable for the resins or balsams, or for the narcotics, in what cases the inhalation of medicines is in place, etc.

According to our opinion, the use of the alkaline-muriatic springs has in numerous cases a real curative influence, not only on the prodromal catarrh, but also on the pulmonary phthisis itself. An effect which is indeed denied by physicians, who are either sceptical or influenced by the older doctrines.

That the use of the water of Ems, Obersalzbrunnen, etc., is contra-indicated in an existing fever, is often misinterpreted. It is not the mineral waters which disagree with the fever, but the traveling to those places of cure, and the "spring promenades." A patient with any considerable fever should be, as we have shown, at home and in bed.

But, since also in the treatment of simple catarrhs, the avoiding of new injurious influences plays a much more important part than the administration of medicines, so is this the case in pulmonary phthisis depending on a catarrhal pneumonia. We would be obliged to advise most phthisical patients to spend the great part of the northern winter indoors, under as uniform a temperature as possible, if these rules were not opposed in very important points to others we have already laid down. We escape the dilemma of being obliged to injure the

patients in one way or the other, if we advise them to avoid the northern winters, and spend that season of the year in a milder climate, where they can pass the greater portion of the day in the open air, without the risk of catching cold, and without breathing a cold, raw atmosphere. That is, according to my view, the only correct value of advising climatic places of *cure*. We must not neglect to urge this sacrifice upon our patients, if their means, etc., allow it, but we must at the same time tell them the truth, in order that they may not believe that the air of the places to which they are sent, contains substances peculiarly curative for their lungs. It is only when the patients themselves know on what it really depends, that they live in Nice, Mentone, Pisa, as well as in Algeria, Cairo, Madeira, carefully enough to expect a good result. If not, it would often be better for them to remain at home. If we act according to these principles, it is self-evident that we should send the patients, even before the commencement of rough weather, during the fall months, to Soden, Badenweiler, Wiesbaden, and especially to the Lake of Geneva, where they can at the same time make use of a grape cure, and, until the beginning of winter, be as well protected as they would be with us in summer. We can risk sending to Nice, Mentone, Pau, etc., only those of our patients who, we feel confident, will remain in-doors during bad weather.

But still, it is better to send people whose means are sufficient, to Algiers, Cairo, Madeira, etc. The advantages which one place has over another are not fully established, and a formula for the different indications of different places for different forms of the disease, etc., would be useless. At any rate, the principal point continues to be, *that the patients, wherever they may be, should live rationally, and remain under the care of a rational and firm physician.*

Against pulmonary phthisis depending upon *primary tuberculosis*, as well as against *tuberculosis which has been developed in the course of a phthisis*, therapeutical agents are indeed powerless, and we are here directed only to a palliative treatment of the most burdensome symptoms. It is a striking fact, that the antipyretic means accomplish but very little against the fever of tuberculosis. If we have recognized that a phthisical patient is tuberculous, we do not send him to Nice, to Cairo, etc., but permit him to spend his last days among his own relations, and die in his own household. This is of course true only of such cases in which the diagnosis of tuberculosis is completely devoid of doubt.

THE END.